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SURGICAL ANATOMY

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THE SURGICAL CLINICS OF NORTH AMERICA

SURGICAL ANATOMY

HENRY N. HARKINS, M.D., *Guest Editor*

Volume 44 — Number 5

OCTOBER, 1964

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SYMPOSIUM ON SURGICAL ANATOMY

Foreword

HENRY N. HARKINS, M.D.

Despite the recent contributions of physiology, chemistry, bacteriology, genetics and other basic sciences, *Surgical Anatomy* still remains the cornerstone of Surgery. This issue includes a series of contributions to this important subject from seven of the ten medical schools on the Pacific Coast of North America and one private clinic, the Mason Clinic of Seattle.

It has been said that, because of the special fellowship requirements in anatomy in Great Britain, British—and to an extent, Canadian—surgeons have excelled those in this country in their knowledge of anatomy. This may still pertain, but to a lesser degree, because in my opinion our surgeons are learning more anatomy, not that the British are knowing less. In medical school education circles some alarmists caution that gross anatomy—and it is gross anatomy applied to Surgery that is commonly defined as *Surgical Anatomy*—is fading out of the curriculum. This is not entirely true and there is still a knowledgeable and inspiring group of teachers of gross anatomy who teach the subject enthusiastically and well. Furthermore, what decrease there may be in quantity of instruction in gross anatomy to medical students is more than made up by an increase in study of the subject during tenure of one of the better surgical residencies.

The authors of this issue have been outstanding students of their particular subjects as related to specific anatomic regions. Some have contributed books in their chosen fields and their present articles represent a sequel, a follow-up, even a "second look," at their subject of interest. All have been contributors to anatomical knowledge, some, such as Sherman (varicose veins), having in the past made distinctive and lasting contribu-

tions in the published literature. Still others, such as Sarjeant, have also made great contributions but have too modestly abstained from putting their ideas in print until they yielded to the persistent urging of the present Guest Editor. In such instances their ideas are printed here and here only.

The subjects covered begin with the head and neck, then move downward to the chest and diaphragm. There is a prolonged pause in the abdomen, still the object of much of the ministrations of the general surgeon. There follow the subjects of the anorectum, hernia (in which chapter Nyhus discourses on his new preperitoneal operation for groin hernia), the pelvis (from the viewpoints of both urology and gynecology), the hand (written by our newest importation from the East as Professor of Surgery at Stanford, Robert A. Chase, to whom we extend welcome), and finally two superb articles on varicose veins and their complications, already alluded to.

It has been a privilege to be the Guest Editor of this volume written by experienced men of stature in Surgery. Their well documented and generously illustrated papers contain specific practical information that should be of value to all practitioners of surgery and to those who look forward to becoming practitioners. These papers also demonstrate that while on the one hand anatomic knowledge usually precedes surgical advances, on the other hand clinical interest in some particular operation or region may cause surgeons to go back to the dissecting room to reappraise the situation. Then, prodded by the urgency for perfecting a practical procedure and armed by clinical experience, they may elucidate new anatomic knowledge which was previously overlooked or not fully understood in its clinical setting.

The Guest Editor wishes to express his appreciation to the contributors, and to his associate, John K. Stevenson, who gave valuable suggestions in the planning of this issue.

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Surgical Anatomy of the Parotid Gland

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Parotid surgery with planned preservation of the facial nerve has been practiced for about 40 years. Prior to that time sacrifice of the nerve was accepted as a surgical necessity during the removal of parotid neoplasms. Increased familiarity with the surgical anatomy of the region now allows parotid surgery in benign disease to be done without loss of seventh cranial nerve function.

A knowledge of the anatomy of the seventh nerve in relationship to the parotid gland is necessary for the performance of biopsy of the gland, superficial lobectomy of benign and noninfiltrative malignant tumors, and for incision and drainage of abscess. In surgical excision of infiltrative cancer, a knowledge of the location of the deep cranial nerves and regional arterial supply is also a necessity.

Parotid Anatomy

The parotid is the largest of the salivary glands. It lies beneath the skin and is surrounded by fascia of varying thickness which is thickest laterally and inferiorly over the gland. Deep extensions of the fascia divide the gland into smaller compartments or lobules.

The outline of the gland is irregular and partially shaped by confines of adjacent structures –the mandible, zygoma, temporal bone and the external ear cartilage. Superiorly, it is bounded by the zygoma, posteriorly by the external auditory canal, mastoid process and sternomastoid muscle. Inferiorly, it extends down to or over the horizontal ramus of the mandible. Anteriorly, the superficial portion extends for varying distances over the masseter muscle. Deeply it extends into the pterygomandibular space lying on the posterior belly of the digastric muscle and styloid process just above it, branches of the carotid artery and jugular vein and in some it extends

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medially as deeply as the pharynx. Many authors feel that the parotid is a bilobed structure.[1, 2, 3] For the surgeon this is of little importance in benign tumors as dissection of the seventh nerve seldom follows a plane of easy separation through the gland. In patients with invasive cancer extending deep to the nerve, this division also is not important as no attempt to separate the lobes is made and the nerve is sacrificed in the interest of curative cancer surgery.

Lymph Node Drainage

Lymphatic drainage from the region of the parotid gland is into the deep jugular lymphatic chain. These are the nodes that commonly become involved with extension of tumor and must be removed in surgery for cancer of the parotid gland. For a detailed description of all the lymphatics of the region, one is referred to the work of Rouviere.[6]

1. *Preauricular Nodes.* One to several nodes are located anterior to the ear and above the fascia. These are most commonly enlarged with facial or scalp inflammation or metastasizing tumors. They are seldom involved from the parotid gland.

2. *Subfascial Nodes.* Inside the parotid sheath lymph nodes are found both in the intraglandular and extraglandular position. Extraglandular lymph nodes lie in the preauricular area and just inferior to the gland. Intraglandular lymphatic tissue varies in size and location. It may be microscopic only or actually nodal in character and of varying size. It is seldom of importance to a surgical dissection. In cancer surgery the lymph nodes lying just below the gland adjacent to the external jugular vein and the sternomastoid muscle are an important metastatic site. These nodes are in continuity with the deep jugular lymphatics and where clinically positive may have to be resected in continuity with this region.

Surgical Anatomy of the Facial Nerve

The facial nerve emerges from the stylomastoid foramen. The foramen lies posterior to the styloid process, deep to the mastoid process and anterior to the origin of the digastric muscle in the mastoid groove of the temporal bone (Fig. 1). It then passes downward, forward and outward, often crossing the styloid process obliquely, and enters the posterior portion of the parotid gland (Fig. 3). With the parotid retracted anteriorly, one can usually visualize 1 to 1.5 cm. of visible nerve (Fig. 2). Shortly after entering the gland, usually at 0.5 to 1 cm., it bifurcates into the temporofacial and cervicofacial divisions (Fig. 4). The branching pattern subsequently is highly variable. For details of the variation of pattern the reader is referred to the work of Davis et al.[2] The branches of the seventh nerve emerge from the gland on the back side of the superficial portion near its anterior border and lie on the masseter muscle. The extreme variability of the branches with their peripheral anastomoses must be appreciated. However, successful preservation of the nerve function is not dependent upon

nerve pattern but identification of the trunk and its branches and avoidance of injury to the nerve in the dissection.

Vessels and Nerves of the Parotid Region

The seventh nerve is the most important structure to trespass the parotid substance. Injury produces the loss of facial expression. The most serious sequela, from a functional standpoint, is an inability to close the eyelids which may lead to exposure damage to the cornea.

There are several possible ways one might injure the seventh nerve causing temporary or permanent damage. It may be cut, ligated, stretched, pinched or dehydrated. Injury may be avoided by detailed knowledge of the anatomy, extreme care in dissection, a blood-free field during the dissection, and prevention of dehydration. Sacrifice of the arteries and veins traversing the gland does not produce harmful effects. They are important primarily in hemostasis. Branches of the external carotid artery and posterior fascial vein traverse the gland.

Parotid Duct

For a detailed description the reader is referred to Davis et al.[2] The parotid duct is of little importance after removal of the superficial lobe or

Fig. 1

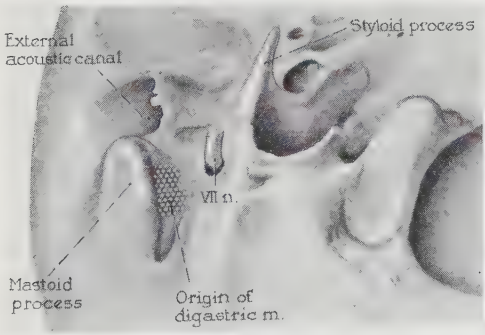
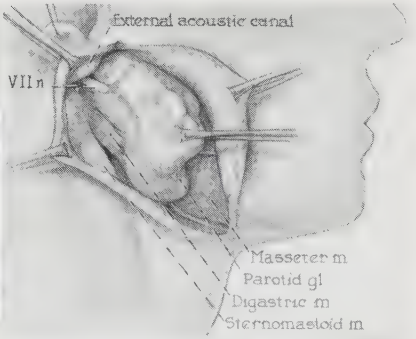


Fig. 2



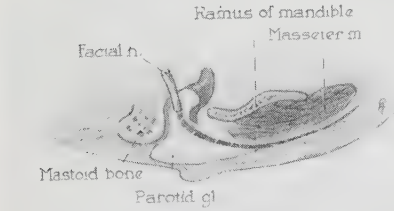


Fig. 3

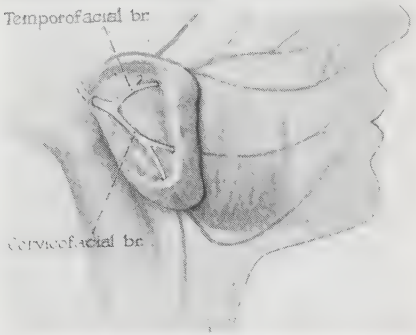


Fig. 4

Figures 1 to 4. Origin and course of the facial nerve. (See text.)

entire gland. If the duct is severed by regional trauma with a normal gland present the placement of a temporary stent of polyethylene tubing through the area of injury may be useful.

Skin Incision—Flaps

For exposure of the parotid gland, the incision is made vertically starting at the zygoma and extending downward 1 cm. anterior to the ear (Fig. 5). Immediately below the ear, the incision should either be extended as a Y-shaped incision behind the ear (Fig. 5), or carried backward 2 to 3 cm. over the mastoid process and then directed obliquely downward and anteriorly beneath the ramus of the mandible. The same incision can be extended forward and be intercepted by the vertical limb of the neck dissection incision when neck dissection in continuity is indicated (Fig. 5). The incision is deepened down to the deep fascia overlying the gland unless the skin is involved by infiltrative tumor. At the level of the fascia, flaps are dissected forward exposing the entire gland. As the seventh nerve lies intraglandular and posterior to the superficial portion of the gland anteriorly, flaps can be dissected without worry of injury to the nerve. To drain a parotid abscess, the flap is dissected. Then a sharp hemostat is introduced through the fascia and spread in the direction of the nerve fibers[4] (Fig. 6).

Isolation of the Seventh Nerve

For benign tumors and small malignant tumors, it is easier to approach the major trunk directly in its fixed location. Where this is not possible it may be necessary to approach the cervical branch and follow it posteriorly to the major trunk. This is accomplished by following up the external jugular vein to the posterior facial vein where the cervical division of the seventh nerve crosses the anterior division of the posterior facial vein (Fig. 7). However, it is usually more simple and less time-consuming to approach the major trunk directly.

After dissection of the flaps the thick facial fascia is opened just anterior to the ear cartilage. With the fascia open the dissection is carried caudal just anterior to the mastoid process and sternomastoid muscle. By blunt dissection spreading in the direction of the nerve this potential space is enlarged. Deep and cephalad to the sternomastoid muscle one can next expose the posterior belly of the digastric muscle. The seventh nerve is found emerging between the posterior belly of the digastric muscle and the bony part of the external ear canal (Fig. 2). Location here is constant. With the gland retracted forward, putting the nerve on a slight stretch, it is readily palpated and may be followed forward and inferiorly to where it dips into the deep parotid substance. The tissue immediately overlying the nerve is separated by blunt dissection with either a fine curved mosquito forceps or fine-pointed scissors exposing the nerve. Fascial strands are cut

and where vessels are exposed they are clamped, cut and ligated. The superficial portion of the gland is mobilized. The nerve should be kept moist with Ringer's solution to prevent dehydration, a common cause of temporary postoperative dysfunction. In deep dissection for the occasional tumefaction occupying the so-called deep lobe of the parotid, it is necessary to do a superficial dissection to define the seventh nerve followed by a deep lobe dissection. The tumor can usually be extracted from below the seventh nerve without the necessity of cutting it. In mixed tumors of the deep lobe, the gland is usually separated by blunt dissection from the surrounding structures at the level of the capsule of the gland. Rarely, the external carotid artery and posterior fascial vein may penetrate the deep lobe requiring ligation and division of these vessels. In large deep lobe tumors an assistant's finger exerting pressure against its protrusion into the pharynx may aid in tumor removal.

Where an infiltrative cancer is suggested by the appearance of the exposed tissue, it is desirable to establish a diagnosis by immediate frozen section before sacrificing the seventh nerve. If metastatic malignancy is

Fig. 5

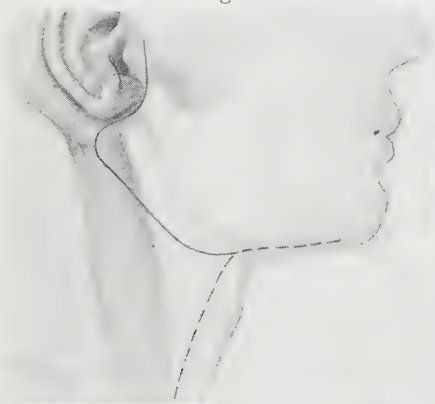
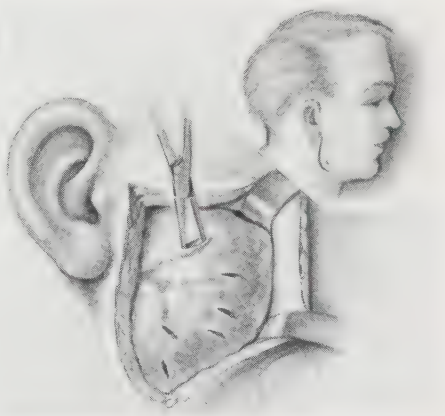


Fig. 6



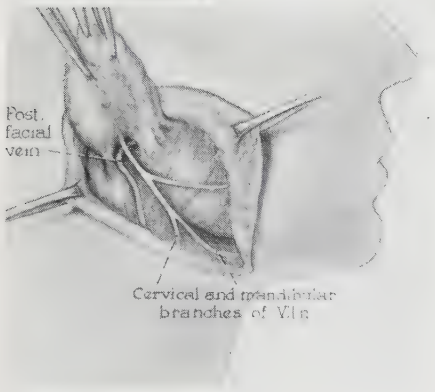


Fig. 7

Figure 5. Incision for exposure of the parotid gland.

Figure 6. Drainage of a parotid abscess.

Figure 7. Isolation of the seventh nerve and its branches.

(For details, see text.)

found in one of the regional nodes, a standard neck dissection in continuity with total parotidectomy and sacrifice of the seventh nerve is accomplished.

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Surgical Anatomy of the Neck

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There is an abundance of detailed information on the applied anatomy of the neck. Rather than attempt to duplicate this material it will be the author's purpose to point out the structures encountered in areas of the neck entered most frequently by the general surgeon. The experience of assisting many generations of surgical residents has taught the author that there are several areas where dissection may be difficult for the uninitiated and where important structures may be damaged. Rather than "surgical anatomy of the neck," this section might well be entitled "how to avoid trouble in the neck."

GENERAL CONSIDERATIONS

The anatomy of the neck is remarkably constant and, once the surgeon gains familiarity with it, important structures may be anticipated, readily identified and preserved. In few surgical fields are clean anatomic dissection, careful hemostasis and wide exposure of more importance, for it is by these measures that operative problems and complications are avoided.

While an earlier generation of surgeons performed marvelous feats with local infiltration or nerve block anesthesia, it is the author's conviction that for adequate exposure and safety, most operations on the neck should now be performed under general anesthesia, preferably with an endotracheal tube in place. Exceptions to this principle may be made for the excision of skin lesions, epidermoid cysts and a few other minor conditions. All too frequently, however, an anticipated "minor" procedure in the neck with local anesthesia suddenly becomes a major problem. The author has witnessed and helped control serious hemorrhage during scalene lymph node biopsies under local anesthesia, has noted spinal accessory nerve damage following excision of lymph nodes in the posterior cervical triangle,

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and has frequently found that removal of a "superficial" lesion leads one into contact with important deep structures.

Positioning of the patient in the head-up or reverse Trendelenburg position is of value. This improves venous drainage and aids in exposure. The slightly increased risk of air embolism if a major vein is entered has thus far been only theoretical. Exposure is also facilitated by extension of the neck by means of a pad or small sandbag beneath the shoulders and by turning the head far to the unaffected side.

Generously long incisions allowing adequate exposure for the task at hand are important. Keloid formation is rather uncommon in the neck and well planned incisions are seldom unsightly. The skin lines of the neck run generally in a transverse fashion and incisions are best when they conform. A useful incision in the upper neck commences below the mastoid and curves downward and forward toward the tip of the thyroid cartilage. A slightly curved transverse incision 2 cm. below and parallel to the body of the mandible gives excellent exposure to the submandibular region and seldom results in a noticeable scar. The standard collar incision for thyroid surgery or an outward extension of this paralleling the clavicle is useful in the lower neck. For major problems and the widest exposure, however, an oblique incision along the anterior margin of the sternocleidomastoid muscle gives maximum safety. This can be carried if need be from the mastoid process to the sternum and horizontal extensions may be made along the mandible and the clavicle from the upper and lower ends. While this oblique incision does not conform to skin lines, it is the safest for extensive procedures and the resulting scar is usually acceptable.

THE DIGASTRIC TRIANGLE (Fig. 1)

This triangle of the neck, bounded by the edge of the mandible and the anterior and posterior bellies of the digastric muscle, must be entered for excision of the submaxillary salivary gland or lymph nodes about it, for exposure of the mandible, and as part of a radical neck dissection or suprahyoid dissection. As the area is approached, usually through a transverse submandibular incision, one must look for the mandibular branch of the facial nerve. This tiny filament courses horizontally beneath the platysma and upon or in the underlying fascial layer, a varying distance of 0.5 to 2 cm. below the mandible. The nerve crosses superficial to the facial artery and accompanying anterior facial vein below the point where they cross the notch of the mandible. A bit posterior to this, a cervical branch of the nerve may be seen extending downward in the neck before the nerve itself disappears upward into the parotid. Anteriorly, before reaching the insertion of the digastric, the nerve curves upward over the edge of the mandible. This nerve is frequently difficult to identify. A helpful maneuver is to observe the corner of the mouth while sweeping the

handle of the scalpel from the midpoint of the mandible downward on the neck. The lip will twitch as the nerve is crossed. Injury to this nerve results in paralysis of the lower lip on the involved side; fortunately in many instances there will be recovery, through anastomosing branches of the facial nerve.

After the mandibular branch has been identified, and by dissection beneath it gently swept up to safety over the mandible, the superior border of the digastric triangle may be defined. A transverse incision is made on the lower free edge of the mandible commencing just back of the insertion of the anterior belly of the digastric. As the incision is carried down to bone, the contents of the triangle are bluntly pushed downward. Near the midpoint of the mandible, the facial artery and anterior facial vein must be divided. After freeing the superior border of the triangle, the anterior belly of the digastric is next exposed by sharp dissection. The submaxillary

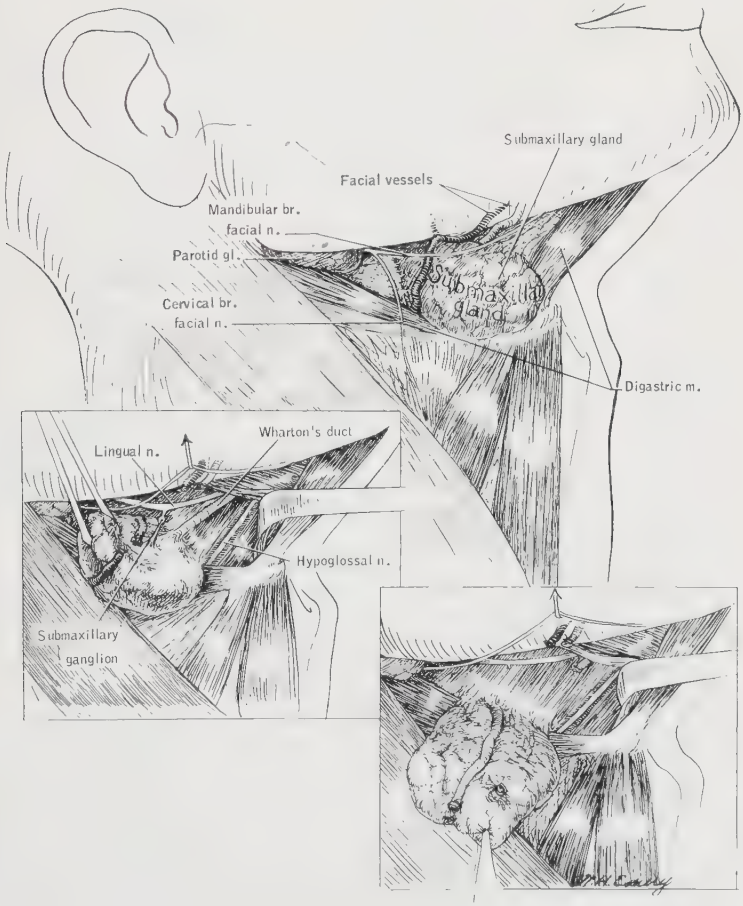

Figure 1. The digastric triangle.

salivary gland frequently bulges out of the triangle over the anterior belly of the digastric, but it is easily elevated as the muscle is followed downward from the mandible to its tendinous portion on the hyoid bone.

The key to successful dissection from this point on is identification of the free posterior edge of the mylohyoid muscle. Failure to locate and retract this anatomical landmark leads to troublesome bleeding and possible injury to important structures. Dissection is commenced anteriorly in the acute angle formed by the mandible and the anterior belly of the digastric. Areolar tissues and one annoying small artery are divided as the mylohyoid is exposed. Dissection is then carried out directly upon the muscle, sweeping areolar tissue, lymph nodes, and an overhanging portion of the submaxillary gland posteriorly. At length a vertical free edge of the muscle is reached and success is at hand. A small retractor is used to retract the mylohyoid forward, exposing the floor of the digastric triangle formed by the hyoglossus. The submaxillary gland is pulled downward; this brings the large lingual nerve into view since it is attached to the gland by small nerve twigs (submaxillary ganglion). When this attachment is divided the lingual nerve again ascends behind the mandible. The submaxillary gland may now be rotated downward and Wharton's duct is identified, extending anteriorly and upward toward the floor of the mouth. The hypoglossal nerve becomes visible coursing obliquely upward and anteriorly upon the hypoglossus muscle. A fairly large lingual vein lies on either side of the hypoglossal nerve and bleeding is avoided by bluntly sweeping the submaxillary gland away from the floor of the triangle.

The contents of the digastric triangle are now pulled posteriorly to complete the dissection as attention is directed to the posterior belly of the digastric. As this muscle is exposed the last remaining important structure is visualized. The facial artery which had been divided above at the mandible, enters the triangle posteriorly and inferiorly as it ascends beneath the posterior belly of the digastric to course along the deep surface of the submaxillary gland. It is a fairly large vessel as it enters the triangle just a short distance from its origin from the external carotid, and it must be carefully secured. Once the artery has been divided, dissection of the digastric triangle can be rapidly completed.

THE STERNOMASTOID OR CAROTID REGION (Fig. 2)

The broad sternocleidomastoid muscle covers a large portion of the lateral cervical region. Beneath it lie the carotid sheath and numerous important structures. A wide variety of surgical conditions require exposure of all or part of this area. These include branchial cleft cysts and sinuses, the important deep jugular lymph nodes, lesions of the carotid artery, neurogenic tumors, deep abscesses and pharyngeal diverticula. This zone

must also be traversed for exposure of the anterior surface of the cervical spine.

The key to successful dissection in this region is wide exposure and then retraction of the anterior margin of the sternocleidomastoid muscle. The outline of the muscle is easily noted by inspection and palpation of the neck. After the platysma and investing fascial layers are incised, the anterior margin of the muscle is isolated for as great a distance as the incision will allow and is then retracted posteriorly. As the fascia over the muscle is incised in the upper third of the neck, care must be taken to avoid injury to the great auricular nerve. This nerve, originating from C-2 and C-3, passes around the posterior border of the sternocleidomastoid and

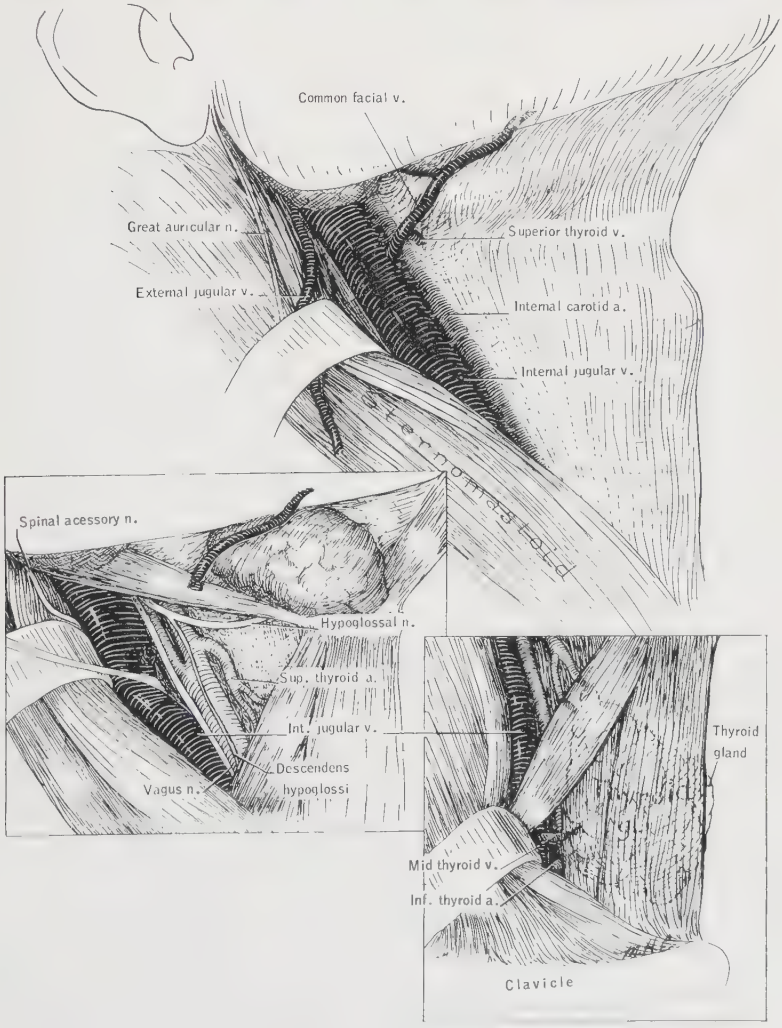

Figure 2. The sternomastoid or carotid region.

curves forward and upward toward the ear, lying close to the anterior margin of the muscle. Injury to the nerve results in an annoying anesthesia of the lobe and posterior surface of the ear. The upper portion of the external jugular vein after its exit from the parotid lies close to the great auricular nerve.

As the upper third of the sternomastoid is retracted the carotid sheath comes into view. This fascial layer is gently spread to expose the internal jugular vein which passes upward to disappear beneath the posterior belly of the digastric muscle. An important group of lymph nodes, the deep jugular nodes, lie along the course of the vein. A node or group of nodes lying upon the vein just inferior to the posterior belly of the digastric (subdigastric nodes) are among the first to be involved by metastatic cancer from the tongue, tonsil or pharynx.

Further exposure in the upper carotid region requires identification and division of the common facial vein, a large tributary of the internal jugular. This vein extends anteriorly and upward from its junction with the jugular toward the submandibular region where it is formed by the confluence of the anterior and posterior facial veins. Just before its union with the jugular, the common facial may be joined by the superior thyroid and lingual veins. When the common facial vein is divided, the internal jugular may be retracted posteriorly for identification of deeper structures.

The common carotid artery is located deep to the internal jugular vein. As the carotid is exposed, the vagus nerve must be identified; it lies on the surface of the carotid and accompanies the internal carotid above the bifurcation. The bifurcation of the artery is somewhat variable in position but generally lies opposite the superior margin of the thyroid cartilage. The superior thyroid artery is readily identified arising anteriorly from the external carotid near the origin of that vessel. The superior thyroid may originate so close to the carotid bifurcation that ligation of the external carotid artery requires preliminary ligation and division of the superior thyroid.

A short distance above the carotid bifurcation the hypoglossal nerve must be identified for safe dissection in this region. This nerve descends from the skull between the internal jugular vein and internal carotid artery. As it emerges beneath the posterior belly of the digastric muscle, the nerve curves forward crossing the external carotid, giving off the descendens hypoglossi as it does so. The hypoglossal nerve is most easily identified at the point where it crosses the external carotid to course anteriorly and again dip beneath the digastric to enter the digastric triangle. It must be gently elevated for wide exposure of the external carotid.

Two important structures lie deep to the carotid bifurcation, one a bit posteriorly and the other extending anteriorly. The posterior deep aspect of the artery is in close relation to the cervical sympathetic chain which lies upon the prevertebral fascia. The superior cervical ganglion may

be identified as a bulbous swelling of the chain a short distance above the carotid bifurcation. Injury to the chain results in Horner's syndrome and unilateral absence of sweating of the face. Extending anteriorly beneath the carotid bifurcation is the superior laryngeal branch of the vagus nerve. This nerve leaves the vagus at the nodose ganglion located just below the jugular foramen. It passes deep to the carotid and curves forward beneath the superior thyroid artery, dividing into two branches to supply the larynx. Injury to this nerve results in partial anesthesia of the larynx, often with aspiration of food and saliva.

One other structure must occasionally be identified in the upper carotid region. This is the spinal accessory nerve which upon leaving the jugular foramen crosses the internal jugular vein obliquely to extend downward and posteriorly in the neck. The nerve is usually obscured in areolar tissue close to the deep posterior surface of the sternocleidomastoid and it gives off a branch to the muscle, about 4 cm. below the mastoid process. It then enters the posterior cervical triangle which it traverses to reach the trapezius. The accessory nerve may be injured in the upper carotid region during excision of a branchial cleft cyst. Such cysts typically lie upon the carotid sheath beneath the upper third of the sternocleidomastoid. The deep posterior surface of the cyst is in close proximity to the accessory nerve.

The lower carotid region has less complex anatomical relations. Exposure is again obtained by identifying and retracting the anterior margin of the sternocleidomastoid muscle. As this is accomplished the omohyoid muscle immediately comes into view as it crosses the carotid sheath obliquely behind the sternomastoid to ascend vertically with the prethyroid muscles. The omohyoid must be divided for adequate exposure. Division should be accomplished with care since the deep surface of the omohyoid lies immediately upon the internal jugular vein. Further exposure is afforded by division of the middle thyroid vein which courses transversely from the thyroid gland to the internal jugular. Once this is accomplished the carotid sheath may be retracted laterally and the larynx retracted medially for ready exposure of the prevertebral fascia, the bodies of the cervical vertebrae, and the esophagus. Only one additional structure, the inferior thyroid artery, crosses this plane rather low in the neck. This vessel emerges beneath the carotid sheath to reach the posterior aspect of the thyroid gland. It must usually be divided for adequate exposure and delivery of a pharyngoesophageal diverticulum. When the artery is divided, the larynx may be rotated to better expose the posterior surface of the thyroid and the esophagus. The recurrent laryngeal nerve can usually be identified entering the larynx just behind the cricothyroid articulation. Blunt dissection can be carried freely upward in the plane between the esophagus and the cervical vertebrae as high as the nasopharynx.

THE SUPRACLAVICULAR REGION AND POSTERIOR TRIANGLE (Fig. 3)

This large compartment of the neck is bound by the middle third of the clavicle, the trapezius, and the posterior margin of the sternocleidomastoid. The prevertebral fascia covering the scalene muscles forms the floor of the triangle; there is direct communication with the mediastinum inferiorly and the axilla laterally. The space must be entered for lesions of the brachial plexus and subclavian artery, for removal of cervical ribs, for lymph node biopsy, and as part of a bloc dissection of the neck.

In approaching this region the only important superficial structure to be encountered is the external jugular vein. This vessel courses beneath the platysma from the midportion of the sternocleidomastoid vertically toward the clavicle where it dips inward to join the subclavian vein. After the external jugular is divided and the investing layer of deep fascia incised the omohyoid muscle is seen to form a prominent landmark. It extends from the posterior corner of the triangle obliquely upward a variable distance above the clavicle to pass beneath the sternocleidomastoid and over the carotid sheath. The point at which the omohyoid emerges beneath the clavicle and the lower border of the trapezius forms the lower posterior boundary of a radical neck dissection. The omohyoid is enveloped by the pretracheal fascia which then extends inferiorly behind the clavicle to enclose the subclavian vein. When this fascial layer is incised one enters a plane in direct communication with the mediastinum and must identify a number of vital structures.

The carotid sheath lies most medially in the supraclavicular region beneath the clavicular head of the sternocleidomastoid, which must be retracted or divided for adequate exposure. The junction of the internal jugular and subclavian veins is usually beneath the clavicle; occasionally, however, the subclavian vein may rise above the clavicle when the neck is extended. Immediately lateral to the jugular vein is the thoracic duct on the left and the right lymphatic duct on the right. These thin-walled lymphatic vessels ascend into the neck from the mediastinum, passing upward behind the carotid artery and jugular vein, and then arch downward to the lateral aspect of each jugular-subclavian junction. A vertically ascending jugular trunk may be seen joining each major lymph vessel and occasionally a subclavian trunk can be seen extending laterally. While ligation of the thoracic duct or right lymphatic duct may be performed with safety, undetected injury to either of these structures is followed by profuse lymph drainage which frequently requires operative treatment.

Lateral to the carotid sheath and major lymphatic ducts is an important group of inferior deep cervical lymph nodes (scalene nodes) which lie grouped in loose areolar and fatty tissue. These nodes communicate with the deep jugular nodes above and serve as drainage areas for the mediastinum and infraclavicular viscera as well. Coursing transversely in this same region are transverse cervical and suprascapular arteries and veins.

These vessels may be the source of troublesome bleeding during radical neck dissection or scalene lymph node biopsy. The arteries originate from the thyrocervical trunk of the subclavian artery or may arise independently from the subclavian. They emerge on the medial edge of the scalenus anticus muscle and curve laterally to cross the lower neck, roughly parallel to the clavicle. Excessive traction on these vessels may avulse them from their subclavian origin.

The scalenus anticus muscle lies beneath the scalene lymph nodes and extends between the subclavian vein and artery to insert on the first rib. The phrenic nerve is easily identified on the surface of the scalenus anticus; it is the only structure that passes in a medial direction as it descends in the neck. The subclavian artery beneath the scalenus anticus may take a

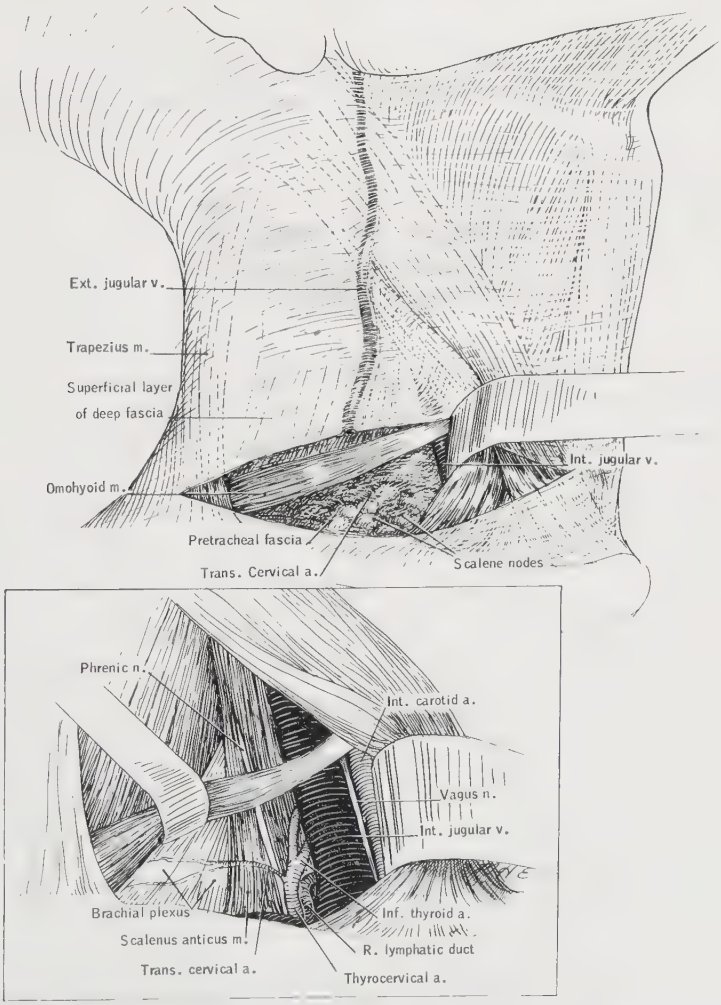

Figure 3. The supraclavicular region and posterior triangle.

surprisingly high curve up into the cervical region before dipping behind the clavicle laterally.

As dissection is carried lateral to the scalene lymph nodes, the brachial plexus may be exposed beneath loose areolar and fatty tissue and beneath the transverse cervical and suprascapular vessels. The trunks of the brachial plexus emerge between the scalenus anticus and medius muscles and course obliquely downward and laterally to pass beneath the clavicle. Both the brachial plexus and subclavian artery may be elevated by a cervical rib; the artery may be compressed by the overlying scalenus anticus in this situation.

SUMMARY

Important structures which may be encountered in many of the common surgical procedures in the neck have been described. Safe surgery in this region depends on a detailed knowledge of the anatomy, wide exposure, and careful hemostasis.

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Surgical Anatomy of the Thyroid and Parathyroid Glands

WILLIAM F. POLLOCK, M.D., F.A.C.S.*

GLANDULAR ANATOMY

The normal thyroid gland weighs about 30 grams in the adult. It usually consists of two lateral lobes connected by an isthmus and lying anterior and lateral to the trachea and esophagus. The gland normally lies just below the cricoid cartilage, but may be somewhat lower in the neck. The lobes vary in size and position; one lobe may be slightly larger or higher than the other. In newborn infants reports of "suffocative goiter" describe the lateral lobes extended so far posteriorly that they almost encircled the trachea and esophagus.[4, 5]

The *lateral lobes* have a broad lower segment and a rather conical apex. The superior pole on each side rises as high as the thyroid cartilage, the lower pole is usually on a level with the fifth tracheal ring. Each lobe is about 5 cm. long, about 3 cm. in greatest width and about 2 to 3 cm. thick. The gland is covered by the skin and platysma, the superficial and deep fascial layers of the neck, the sternocleidomastoid, sternohyoid and sternothyroid muscles. Beneath the muscle layers, it is covered by the pretracheal layer of the deep cervical fascia. Behind it are the parathyroid glands, the trachea and esophagus, the blood vessels and the nerve supply.

The *isthmus* connects the lower part of the lateral lobes; it is about 1.3 cm. in breadth and covers the second and third rings of the trachea. A *pyramidal lobe* may arise from the isthmus or from the adjacent portion of either lobe, more commonly from the left. It rises as high as the hyoid bone in some patients and may be continuous with a thyroglossal duct or may be detached from it. From the upper border of the isthmus, or from a pyramidal lobe to the hyoid bone, there may be a fibrous sheet which is occasionally muscular. When it is muscular, it is called the *levator glandulae thyroideae*. It is sometimes called the "suspensory ligament" of the thyroid

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and is of surgical interest because dividing it gives greater access to the upper pole.

SURGICAL ASPECTS OF THYROID EMBRYOLOGY

The bulk of the thyroid gland is derived from the midventral floor of the embryonic pharynx. As the primitive branchial clefts separate out, a protrusion develops from the floor of the pharynx at the level of the first branchial pouches. By the fourth week of embryonal development this diverticulum becomes an elongated sac, attached to the floor of the pharynx by a tubular neck known as the *thyroglossal duct.* It connects the primitive thyroid structure with the base of the tongue, which is being formed at the same time. The origin of the diverticulum is marked by the foramen cecum, a permanent dimple in the surface of the tongue at the apex of the V-shaped ridge between the filiform papillae anteriorly and the broader vallate papillae on the back of the tongue. The hyoid bone embraces the base of the tongue, hence the invariable relationship of the thyroglossal duct to the midpoint of the hyoid bone and the tongue.

Normally the thyroglossal duct atrophies during the sixth week of embryonal development. Persistence of the duct and its opening into the mouth at the foramen cecum explain the common occurrence of infection in thyroglossal duct cysts. There is no normal connection to the skin; sinus formation is due to infection and external drainage. Simultaneously with the atrophy of the thyroglossal duct, the diverticular thyroid structure becomes bilobed and descends to its ultimate position in the neck, with a lobe on each side of the trachea.

Some textbooks of anatomy state that the thyroid gland arises from three primordia, the main structure arising from the floor of the pharynx and a pair of small lateral diverticula derived from an area at the junction of the *fourth* pharyngeal pouch and the pharynx, known as the *ultimobranchial body.* There are two concepts of whether the ultimobranchial bodies contribute to the adult thyroid gland. One school feels that they are "lateral" thyroid primordia and become an integral part of the completed gland. Others feel that the ultimobranchial bodies degenerate and form no part of the final structure.

Attempts have been made to resolve this controversy in the experimental animal. When the part of the pharyngeal pouch that gives rise to the thymus, parathyroid and ultimobranchial body is transplanted, the graft shows thymic and parathyroid tissue, but no thyroid tissue. When the pharyngeal floor in the area of the thyroid anlage is transplanted, either before or after it appears as a diverticulum, thyroid tissue is regularly identified in the successful transplants. Apparently the ultimobranchial bodies in the experimental animal (mouse) do not form thyroid tissue.

The histologic appearance of thyroid tissue is not always related to

its clinical behavior. The clinical syndrome of "lateral aberrant thyroid carcinoma" is defined as thyroid carcinoma in structures lateral to the great vessels of the neck, *with no palpable abnormality of the thyroid gland.* The lack of a palpable primary tumor led to the original misconception. Many recent studies have shown that the primary focus of cancer will invariably be found if sought with sufficient diligence. Primary tumors as small as 1 mm. in diameter have been demonstrated by serial sections of the entire gland.[7]

"Lateral aberrant thyroid carcinomas" are really metastases to lymph nodes, although they may replace the lymphoid tissue so completely that the primary structure is hard to recognize. Some of these thyroid tissue deposits look surprisingly benign on histologic examination, but there is never any normal thyroid tissue lateral to the great vessels of the neck. Thyroid cancer appearing lateral to these vessels does not mean that there are multiple embryonic origins of the thyroid gland.

There are many examples of anomalous growth or displacement of tissue within the planes of the neck during development of the thyroid primordia, e.g., lingual thyroid, pyramidal lobes, absent isthmus, absent lobes and similar variations. Thyroid tissue has been demonstrated in the larynx and trachea and in remote sites, e.g., brain, pleura, pericardium, skeleton or ovary.

Struma ovarii is due to teratoma formation or tissue metaplasia and is not related to normal thyroid tissue in the neck. Thyroid tissue within the walls of the larynx or trachea is probably an embryologic abnormality in which the thyroid anlage is partially trapped by the other developing structures of the tracheoesophageal complex.

The occurrence of normal appearing thyroid tissue implants in such remote areas as the brain, pleura and spine was formerly called "benign metastasizing adenoma." The benign histologic appearance is misleading; they are metastatic from a primary cancer in the thyroid, probably by way of the vertebral vein channels.[1] An embryologic origin for remote thyroid implants must be discounted in all instances without convincing proof that they are not metastatic cancer.

The presence of part or all of the thyroid gland in the mediastinum is not due to an embryological anomaly. The incidence of *substernal goiter* closely parallels the incidence of goiter generally, and a goiter in the chest is simply an extension from the thyroid in the neck. It is unusual for the entire thyroid gland to lie below the thoracic inlet; it is rare for an intrathoracic goiter to lie below the level of the aortic arch. The vast majority of intrathoracic goiters are in the anterior mediastinum, but occasional cases in the posterior mediastinum have been reported. The surgical significance of these facts is that the blood supply still arises in the neck and ultimately the recurrent nerves must be dealt with in the neck. Almost all cases of substernal goiter can be approached easier through the neck than through the chest. If the goiter within the chest is too large to permit

intact delivery through the upper thoracic inlet, it is safe to morcellate it and remove it piecemeal from the chest into the neck.

In the eighth week of embryologic development small cavities appear in the thyroid tissue and they expand and proliferate as colloid appears in the follicles. By the fifth month of fetal life the formation of follicles is completed and thereafter no new follicles are formed; any further development occurs by the budding and division of existing follicles.

BLOOD AND LYMPHATIC SUPPLY

The arteries supplying the thyroid gland are the superior and inferior thyroid arteries and an additional branch in about 10 per cent of patients called the *thyroidea ima*. This vessel arises from the innominate artery or directly from the arch of the aorta and ascends on the front of the trachea to enter the lower border of the isthmus or of one of the lobes.

The veins form a plexus on the surface of the gland and the front of the trachea, from which the *superior, middle and inferior thyroid veins* arise. The superior and the middle thyroid veins normally end in the internal jugular, the inferior joins the innominate veins. The middle vein is absent in about one-half of the patients.

The common carotid artery bifurcates at the level of the thyroid cartilage and the internal carotid artery has no branches in the neck. The *superior thyroid artery* is the first branch to arise from the external carotid, but in a small percentage of cases, it may arise directly from the common carotid. As it descends to the superior pole of the thyroid gland it lies in intimate association with the branches of the superior laryngeal nerve. After alleged "total" thyroidectomies radioiodine scintigrams often reveal residual thyroid tissue at the upper pole. Complete removal of the upper pole demands exposure of the superior thyroid artery as it arises from the external carotid. This is technically difficult in some patients and may require partial division of the sternothyroid muscle attachment to the thyroid cartilage.

The superior thyroid arteries pass downward on the anterior and medial surface of the upper poles and divide to embrace the thyroid, giving off an anastomotic connection behind the gland to the inferior thyroid artery. Superior thyroid arteries from both sides communicate across the isthmus.

Near the upper pole of the gland, the superior thyroid artery gives off a superior laryngeal branch which accompanies the superior laryngeal nerve across the thyrohyoid membrane to enter the larynx. There may also be a small *cricothyroid artery*, which runs on the cricothyroid membrane close to the lower border of the thyroid cartilage.

The *inferior thyroid artery* is more variable in its course than the superior. The inferior thyroid artery usually arises from the thyrocervical

trunk, which is in turn a branch of the subclavian. The vertebral artery, the thyrocervical trunk and internal mammary (internal thoracic) artery arise close together from the subclavian artery near the border of the anterior scalene muscle. There is a 1.5 to 2.5 cm. distance from the takeoff of the subclavian artery to the first of these three branches, which arise in the order named.

The thyrocervical trunk in turn divides almost immediately into three branches: the inferior thyroid artery, the transverse scapular, and the transverse cervical. The inferior thyroid artery passes upward in front of the vertebral artery and the longus colli muscles and then courses medially *behind* the carotid sheath and the sympathetic trunk, so that the middle cervical ganglion of the cervical sympathetics rests upon this vessel. The artery divides at the lower border of the thyroid gland to supply the posterior and inferior parts of the gland; one branch runs upward on the posterior aspect of the gland to anastomose with the branch from the superior thyroid artery.

It has been said that if there were no such operation as tracheostomy the *thyroidea ima artery* would never have been named; it would simply be a variation of the inferior thyroid artery. The thyroidea ima usually arises from the innominate artery, but it may arise from the common carotid or the subclavian on either side, from the aortic arch directly or from the internal mammary. The vessel arises more commonly on the right and ascends to the lower border of the isthmus of the thyroid along the midline of the trachea, hence its importance in tracheostomy. Depending on what definition is used to name it, its incidence varies from 1.5 to 12 per cent

The *lymphatic vessels* that drain the thyroid gland accompany the arterial blood supply. The superior lymphatic channels drain the upper border of the isthmus and the upper surface of the lobes. The inferior channels drain the major part of the isthmus and the lower part of the lateral lobes. These lymphatic channels empty into the adjacent deep cervical nodes, upper and lower, and into the supraclavicular nodes. The majority of the lymphatic channels from the thyroid end in lymph nodes, but a significant number seem to connect directly to the veins without passing through a node.

The lymphatic vessels and nodes become of interest to surgeons in the treatment of metastatic thyroid cancer. One histologic criterion in the diagnosis of thyroid cancer is invasion of blood vessels, although some authors restrict this to invasion of vessels large enough to have an elastic (media) layer. Blood vessel invasion obviously complicates any plan of resection of lymphatic metastases. Extensions to the thyroid veins and thence into the vertebral veins and to the spine, to the lungs and to retroperitoneal and cranial areas are well known clinical forms of metastatic thyroid cancer.[1]

Surgeons debate endlessly over the question of whether lymph node dissections should be done routinely, done only in cases showing metastases,

or not done at all. The anatomic features of the lymphatic drainage complicate this decision enormously. The standard "radical" neck dissection includes the homolateral jugular vein, the sternocleidomastoid and omohyoid muscles, the smaller regional vessels and the deep and superficial lymph nodes in the lateral and posterior cervical areas, but does *not* include some midline lymph nodes to which thyroid cancer is very apt to spread.

The midline lymph node near the isthmus is so often involved in thyroid cancer it has been called the "Delphian" node, from the oracle at Delphi who predicted things to come. Thyroid cancer often extends downward in the midline to the "thyrothymic omentum" in the upper chest. Some authors advocate removal of these mediastinal nodes as far down as the base of the heart in certain cases.[2, 3] The midline lymphatic metastases in the mediastinum are approached by splitting the sternum, with some possible attendant complications from pleural injury, bone infection, etc. Although the midline lymph nodes are a primary drainage area, the relative inaccessibility of the mediastinal nodes has deterred many surgeons from undertaking wide resection of them.

The lymphatic and vascular channels within the gland itself have also led to surgical controversy. Proponents of *total* thyroidectomy for cancer argue that intrathyroid metastases will occur in 10 to 20 per cent of patients. Opponents of routine total thyroidectomy contend that contralateral spread is uncommon and that the increased risk to the parathyroids and recurrent nerves cannot be justified by any improvement in the survival rate.

NERVE SUPPLY (Figs. 1 and 2)

The skin of the neck in the region of the thyroid gland is innervated from the cervical plexus, chiefly by the superficial cervical nerve. The platysma muscle, immediately beneath the skin, is innervated by the cervical branch of the facial (seventh cranial) nerve. This muscle layer affords the best cleavage plane from the deep cervical fascia for elevation of the skin flaps. The strap muscles are innervated by the descending branch of the twelfth cranial nerve (hypoglossal) united with branches of the cervical plexus to form the ansa hypoglossi. This nerve descends along the lateral border of the sternothyroid and sternohyoid muscles and passes between them to end in the characteristic "goose foot" pattern. Some surgeons clamp and cut the sternothyroid and sternohyoid muscles to get exposure of the gland. There are nerve branches both high and low on the bellies of these muscles, so that if they are cut across, it should be near their midpoint.

It is usually not necessary to cut the muscles to expose the thyroid; they can be separated in the midline and retracted. It may be advantageous to retract the sternohyoid from the midline and then retract the sterno-

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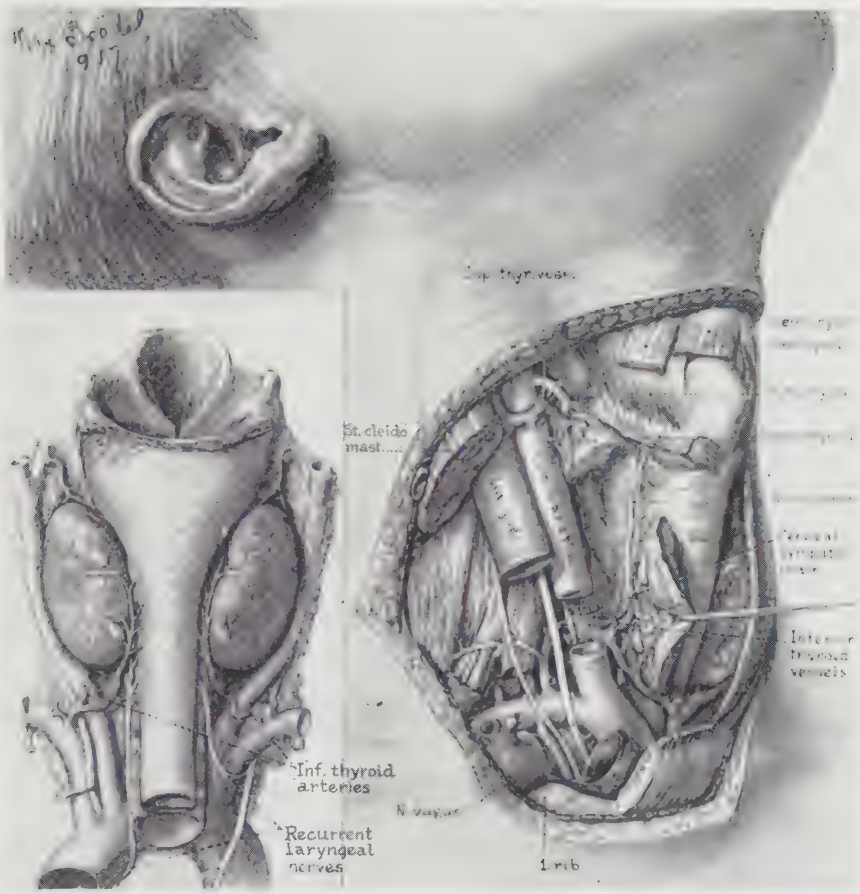

Fig. 1. Fig. 2.

Figure 1. The structures viewed from behind, showing the right recurrent nerve coursing around the innominate-subclavian and its relation to the inferior thyroid artery, which is shown coursing behind the carotid and supplying the parathyroids and then forming the anastomotic plexus with the superior thyroid artery. On the left the origin of the superior thyroid from the external carotid is shown; also the relationship of the recurrent nerve superficial to the vessels. This illustrates how the nerve can be pulled forward as the lobe is rotated anteriorly to secure the blood supply on the posterior aspect of the lobe. (Max Brodel drawing. Property of Department of Art as Applied to Medicine, The Johns Hopkins School of Medicine.)

Figure 2. The origin of the inferior thyroid from the thyrocervical trunk. Its passage behind the common carotid artery and the cervical sympathetic chain is shown and in this example the artery lies anterior to the nerve, contrasting with Figure 1 in which the nerve is anterior to the artery. The superior and inferior arteries and veins are shown, as well as the relative inaccessibility of the superior thyroid artery origin from the external carotid. The cut end of the omohyoid muscle can be seen near the lower edge of the dissection. This muscle's course places it lateral to the usual thyroid exposure. (Max Brödel drawing. Property of Department of Art as Applied to Medicine, The Johns Hopkins School of Medicine.)

thyroid as a separate structure. This maneuver will reveal the ansa hypoglossi coursing inward on the anterior surface of the deeper muscle. The sternocleidomastoid and omohyoid muscles are lateral to the standard thyroid approach, though they become anatomically important in dissections of the cervical lymph nodes.

The larynx is supplied by branches of the vagus nerves. The *superior laryngeal nerve* courses near the superior thyroid and laryngeal arteries. The *inferior* or *recurrent nerves* are more subtle and more significant clinically. The right recurrent nerve hooks backward around the subclavian artery and then courses upward beside the carotid artery to the tracheoesophageal groove. It enters the area of the thyroid gland in some intimate relationship with the inferior thyroid artery. On the left, the nerve hooks backward around the aortic arch at the ligamentum arteriosum, which is the remnant of the ductus arteriosus. It then courses upward in a manner similar to that on the right and passes into a similar relationship, not necessarily symmetrical, to the inferior thyroid artery.

Many surgeons believe that the recurrent nerves are so deep in the tracheoesophageal groove that they can be avoided by staying lateral to this area. This is by no means the case. The nerve may run as much as a centimeter lateral to this groove, the right side being more apt to do this than the left. The relationships of the recurrent nerve are best considered with reference to the inferior thyroid artery. Depending upon how far lateral it lies, the nerve may cross the inferior thyroid before the artery divides, as it divides, or after it has divided. The nerve on either side may pass in front of, behind, or between the branches of this artery. Many descriptions of this anatomic relationship have been published and it is certain that no consistent pattern or overwhelming preponderance has been found. Over 20 different types of nerve-artery relationship have been classified and the relationship is symmetrical on both sides in only a small percentage of cases.

On the *right* side, the nerve passes between branches of the artery in about 50 per cent, in front of the artery in 25 per cent and behind it in 25 per cent of cases. On the *left* side the nerve passes behind the artery in 50 per cent, between branches in 30 per cent and in front of the artery in 20 per cent of the cases. If the nerve runs anterior to any branches of the artery, the vessels can pull the nerve forward as the lobe is rotated medially. Displacement of the thyroid lobe offers no guarantee that the recurrent nerve will be protected.

In the majority of patients the recurrent nerve divides before it enters the larynx. This division helps to explain differential injury to the "abductor" or "adductor" functions and the varying patterns of vocal cord function resulting from damage to the recurrent nerves.

There are two methods proposed to avoid injury to the recurrent nerve. One school advocates routine exposure of the nerve, while others say that the nerves are often injured in a determined search for them.

They contend that the nerve is usually visualized *below* the area where injury is most apt to occur, i.e., in ligating the branches of the inferior thyroid artery near the gland. There is no true thyroid capsule, but many surgeons dissect in the so-called "surgical capsule," a distinct layer of areolar tissue lateral and posterior to the gland. This layer should serve as a protecting mantle for the recurrent nerves and parathyroid glands.

The incidence of nerve injury is higher in operations done for cancer, or for nodular or recurrent goiters than it is for operations on diffuse goiters. A posterior shell of thyroid tissue is usually left behind when operating on diffuse goiters to protect the nerves and parathyroid glands. Efforts to remove every milligram of thyroid tissue in the neck will damage the nerves and parathyroids in a substantial percentage of cases. Exposure of the nerve is no guarantee that it will not be damaged. The mere handling of it, or dissection in close proximity to it, can cause temporary loss of function for days to weeks. Local anesthesia is sometimes used in thyroidectomy to permit active voice monitoring during this dissection.

After-Effects of Nerve Injury

Injury to the *superior laryngeal nerve* has no dramatic or dangerous after-effects, so that its true frequency is probably not known. Injury to the superior laryngeal nerve causes paralysis of only one muscle, the cricothyroid. Hoarseness is not a common complaint, but inability to achieve high tones and voice fatigue are common results of bilateral superior nerve injury. Sensory function is impaired in the interior of the larynx. The autonomic control of mucus secretion may be disrupted, since the sympathetic outflow from the cervical ganglia courses with the superior laryngeal nerves. This leads to a sensation of mucus accumulation and frequent clearing of the throat.

Bilateral loss of superior laryngeal nerve function does not cause dyspnea or any interference with swallowing or breathing. Upon laryngoscopic examination, there may be no obvious alteration in the appearance of the resting cords. The paralyzed cricothyroids cannot tense the cords, but this may be concealed by the other intrinsic muscles, which take up the slack on attempted phonation.

Injuries to one or both *recurrent (inferior) laryngeal nerves* can produce a surprising variety of clinical situations. Some of the differences in the position and function of the vocal cords are not easily demonstrated and may not be recognized except by experienced examiners. Part of this problem lies in evaluating the relationship of the cord to the anatomic midline of the glottis. "Fixed" or "paralyzed" vocal cords may be straight or bowed. The "cadaveric" position is an example of paralyzed bowed cords.

The larynx performs three functions: (1) vocalization (pitch and loudness) involving all the laryngeal muscles except the posterior cricoarytenoid: (2) respiration; opening the glottis by action of the posterior cricoarytenoid,

and (3) protection of the airway during swallowing, involving all the muscles *except* the cricothyroid and the posterior cricoarytenoid.

Intelligible speech is made only with the tongue and lips. Patients attempting suicide, who have cut their throat at a level just above the larynx, but not deeply enough to be fatal or to injure the vagus nerves, have provided unusual demonstrations of laryngeal function. With the larynx exposed by such a cut, in a conscious patient trying to talk, the cords can be seen to adduct and abduct very rapidly, with the production of a buzzing sound, varying in pitch and intensity, but totally unintelligible. It sounds like a vibrating pair of reeds, as in some woodwind instruments. When the head is brought forward, so that the column of vibrating air enters the mouth, the tongue and lips form intelligible speech. Laryngectomized patients who have learned the esophageal voice demonstrate the functions of the lips and tongue as opposed to those of the larynx.

If one recurrent nerve is damaged near the thyroid gland, there may be some dyspnea upon exertion, but little hoarseness, since the opposite cord can cross the midline to meet the paralyzed one. If both recurrent nerves are damaged, the voice will be weak and hoarse, but there is little dyspnea while resting. The hoarseness arises from inability to adduct the cords. By tensing the cricothyroid muscles, which are supplied by the superior laryngeal nerves, the patient's voice may improve, but his airway will then be narrowed and dyspnea will result, especially on exertion.

The vocal cord derangement resulting from recurrent nerve injuries depends on how much of the nerve supply is damaged, how long after the damage the loss is evaluated and the state of the larynx before the injury occurred. If the left recurrent nerve is damaged in an operation on the ductus arteriosus or aortic arch, the left cord adducts normally, yet injury to the same nerve near the larynx results in loss of adduction. Some patients have transient changes in vocal cord function after thyroidectomy, appearing immediately and clearing spontaneously after days or weeks. The delay in function is usually due to edema from the surgical manipulation.

LINGUAL THYROID AND THYROGLOSSAL DUCT CYSTS

The thyroid arises from the pharyngeal floor in the same area as the hyoid bone and base of the tongue. If the thyroid tissue completes its development but fails to descend and remains entirely embedded in the tongue, this developmental anomaly is known as lingual thyroid. In three-fourths of these patients, the lingual thyroid represents the *only* thyroid tissue they have. For this reason, it is essential to determine whether any other thyroid tissue is present before removing a lingual thyroid. With radioiodine techniques, it is possible to determine (*a*) whether the intra-

lingual mass is functioning thyroid tissue and (*b*) whether there is any other functioning thyroid tissue elsewhere in the neck.

Lingual thyroids have been approached from within the mouth, through the suprahyoid region from the side, or preferably by entering the base of the tongue from below. A tracheostomy has been necessary to guarantee the airway during and after this dissection in many cases.

Ectopic thyroid tissue in the tongue or neck always arises as a midline mass and has an intimate relationship to the hyoid bone and tongue for the embryologic reasons noted earlier. Most surgeons prefer the suprahyoid approach for complete removal of lingual thyroid or thyroglossal duct cysts. Usually the thyroglossal duct cannot be completely removed without removing the central portion of the hyoid bone. No disability results from the removal of the center of this bone, and the cut ends of the hyoid can be left free in the muscle substance; they need not be reunited.

The thyroid epithelium in any ectopic location is capable of producing every form of thyroid disease—thyroiditis, carcinoma, adenomas of all cellular types, and hyperthyroidism. Almost every case of lingual thyroid or thyroglossal duct cyst treated by radioactive iodine has subsequently come to excision because of persistence of the mass, dysfunction, etc. There is a substantial risk of cancer in ectopic thyroid tissue. Ectopic thyroid tissue becomes symptomatic during adolescence in about one-half of the cases. Thyroid tissue has no capsule and there is no cleavage plane between any form of ectopic thyroid tissue and the surrounding muscles.

Autotransplantation of the excised lingual thyroid tissue has been attempted many times and has met with varying degrees of success.[6] The situation is clearly favorable for this attempt. The lingual tissue usually represents the only thyroid tissue the patient has, so that its removal creates a demand, which some believe essential to success in transplantation. It is autologous, it is highly vascular, it requires no nerve supply for its function and it is not essential to life if it fails, since oral thyroid in any amount is readily available. Thin tissue slices (1 to 2 mm. thick) are optimal for success in autotransplantation and the recipient site is relatively unimportant. Several sites should be used, so that one graft can survive if infection destroys the others. There is always a lag of several weeks between graft placement and detectable function. Withholding oral thyroid medication while awaiting function of the graft is logical. In patients with functioning autografts, the administration of oral thyroid will decrease the pituitary stimulation of the graft. The graft's function fluctuates with the amount of supplemental thyroid hormone available.

PARATHYROIDS

The origin of the parathyroids from the third and fourth pharyngeal pouches is well documented and apparently almost invariable. There is

great uncertainty about how many parathyroids may be considered the "normal" number. There are usually at least four, one placed posteriorly near the upper and lower poles of the lateral thyroid lobes on each side. There may be more than one in each of these sites and as many as ten areas of histologic parathyroid tissue have been described, scattered in various locations around the thyroid gland and thymus. True ectopia of parathyroid tissue can occur, with functioning parathyroid tissue lying high in the neck, in areas independent of the thyroid gland, or in the mediastinum in association with the thymus.

The blood supply to the parathyroid glands is basically that of the thyroid gland. This blood supply is so rich that if both superior and inferior thyroid arteries and the thyroidea ima were all ligated, no necrosis of the thyroid or of the parathyroids would ensue. The anastomotic connections to the vessels supplying the trachea, esophagus, pharynx, and larynx are so extensive that no alteration of function ensues from peripheral ligation of the principal arterial supply. Many years ago, "staged" ligation of the superior pole vessels for severe hyperthyroidism was used; it is now known that this operation had no discernible effect on the function of the thyroid gland.

The parathyroids are small, elongated, golden brown bodies lying on the posterior part of the thyroid gland and in the areolar tissue near it, the so-called "surgical" capsule. Identification of the parathyroids demands absolute hemostasis. Bleeding into or around them can obscure them so completely that their precise identification is impossible. In this area of surgical endeavor, careful, meticulous dissection in an absolutely dry field is essential for success. The parathyroids are concerned with phosphorus and calcium metabolism and are of surgical interest because of hyperfunction or because of deficiency. Hyperfunction occurs from functioning adenomas or from clear cell hyperplasia with approximately equal frequency. Loss of parathyroid function is usually due to surgical injury or removal, although idiopathic hypoparathyroidism also occurs.

Some attempts have been made to correlate the incidence of parathyroid deficiency after thyroid operations with where the inferior thyroid artery was ligated (*a*) as a single vessel lateral to the thyroid lobe or (*b*) by multiple ligation of small vessels in intimate proximity to the gland. There is no clear advantage from either technique. If serial sections are done of the entire thyroid specimen submitted for pathologic examination, some histologic evidence of parathyroid tissue will be found in a remarkably high number of cases. Partial or transient parathyroid deficiency during the immediate postoperative period develops after thyroidectomy in about one-fourth of the cases.

Total and permanent parathyroid deprivation is rare and is usually related to efforts at total thyroidectomy for treatment of thyroid cancer. In the present state of our knowledge it is not possible to correct total

parathyroid deprivation by surgical transplantation, but medical supportive methods are adequate.

Surgeons are naturally interested in avoiding this complication and again there are two schools of thought. Routine identification may protect some of the parathyroid glands, but the majority opinion seems to lie with protecting them by staying away from them. The damage usually occurs in relation to efforts to secure the blood supply. Assuming that it is impossible to compromise the parathyroid glands by depriving them of their *remote* blood supply, the damage must come from ligating the blood supply too close to them, or actually removing them.

If the inferior thyroid arteries are ligated as isolated structures, the parathyroids may be safer, but the risk of damage to a recurrent nerve in that area may be higher. If this problem is avoided by multiple vessel ligation in very close proximity to the gland, the risk of injury to the parathyroids may be greater. Actual removal of parathyroid tissue occurs more commonly than is generally recognized.

There is no certain anatomic guide to the parathyroid glands, or any certain way of determining whether there are four or more or less. In the cases with hyperfunctioning adenomas of the parathyroids, the remaining parathyroid glands may be smaller than normal, from "disuse atrophy." Every surgeon should get to know the appearance and size of the normal parathyroid gland, so that he will recognize variations in a parathyroid gland when he sees one, regardless of where it may lie.

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The Anatomical Basis for Surgical Correction of Carotid Insufficiency

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Atherosclerosis of the cervical arteries is common in the elderly and is probably more frequent and more extensive than occlusive disease of the cerebral arteries.[8, 13, 17, 24] Martin and his co-workers[17] found that 11 per cent of unselected patients over 50 years of age had total occlusion of at least one artery in the neck. In 40 per cent of these patients severe stenosis had occurred in one or more arteries to such an extent that less than one-half of the lumen remained. In a study of 83 autopsies in patients with clinical evidence of cerebral vascular disease, Hutchinson and Yates[13] found that in 40 of them one-half or more of the lumen of either a carotid or a vertebral artery, or both, was obliterated. Gurdjian and his co-workers[11] found the incidence of cervical arterial disease as high as 41 per cent in 720 patients presenting with signs of cerebrovascular insufficiency.

The integrity of the cervical arterial supply, that is to say the total blood supply of all four cervical arteries, profoundly influences cerebrovascular sufficiency. Obviously other factors are involved.

THE COLLATERAL CIRCULATION

The collateral circulation assumes a paramount role in the presence of cervical arterial occlusive disease. Most important in this regard is the circle of Willis; when it is normally formed, complete occlusion of one, sometimes two and, rarely, three of the major arteries in the neck may be unheralded.[5, 7, 10] Figures 1 and 2 are examples of effective collateral circulation through an intact circle of Willis.

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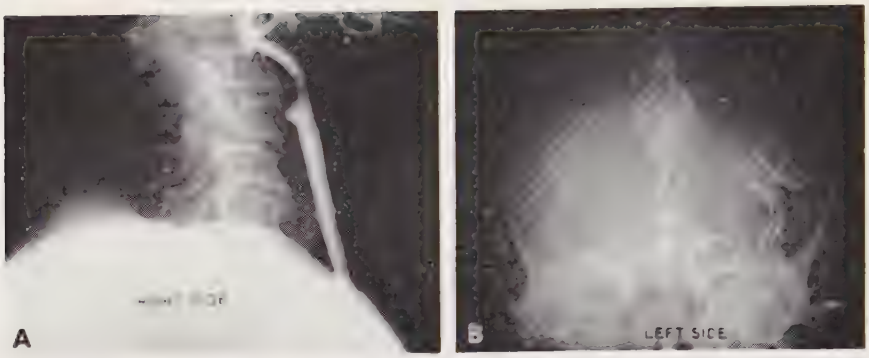

Figure 1. *A*, Right carotid arteriogram demonstrating total occlusion of the internal carotid artery.

B, Left carotid arteriogram showing excellent cross circulation through an intact anterior communicating component of the circle of Willis.

Figure 2. Retrograde brachial arteriogram showing good fill of the internal carotid artery via the posterior communicating artery in a 58 year old woman with bilateral cervical internal carotid artery occlusion.

Unfortunately, the circle of Willis cannot always be relied upon as a major anastomotic pathway in man since its classic configuration may occur in less than one-half of the population. Alpers and his co-workers[13] found the textbook description of the circle of Willis present in only 52.3 per cent of 350 brains at autopsy. Variations of the basal cerebral arteries, including the circle of Willis, were found in 65 per cent of Kameyama and Okinaka's series of 400 elderly patients.[14]

Case I. A 77 year old man had, for 6 months, transient attacks of lightheadedness and blurred vision. One week prior to admission to the Virginia Mason Hospital he suddenly developed moderate weakness on the right side which persisted.

He presented with a mild dysphasia, a right central, facial weakness, and a mild right hemiparesis. His blood pressure was 200/100.

Ancillary studies were not helpful except for an electrocardiogram which showed ventricular ischemia, secondary to arteriosclerotic heart disease. Left carotid arteriography demonstrated almost complete occlusion of the internal carotid artery (Fig. 3, *A*). A right carotid arteriogram showed an intact carotid bifurcation in the neck. Much to our consternation, both anterior cerebral arteries filled from the left (Fig. 3, *B*) and were totally dependent upon and almost completely occluded the internal carotid artery.

Endarterectomy was carried out soon afterwards under general anesthesia with induced hypertension during the period of vascular occlusion. Eleven minutes following the onset of occlusion, respirations ceased. Despite the fact that the arteriot-

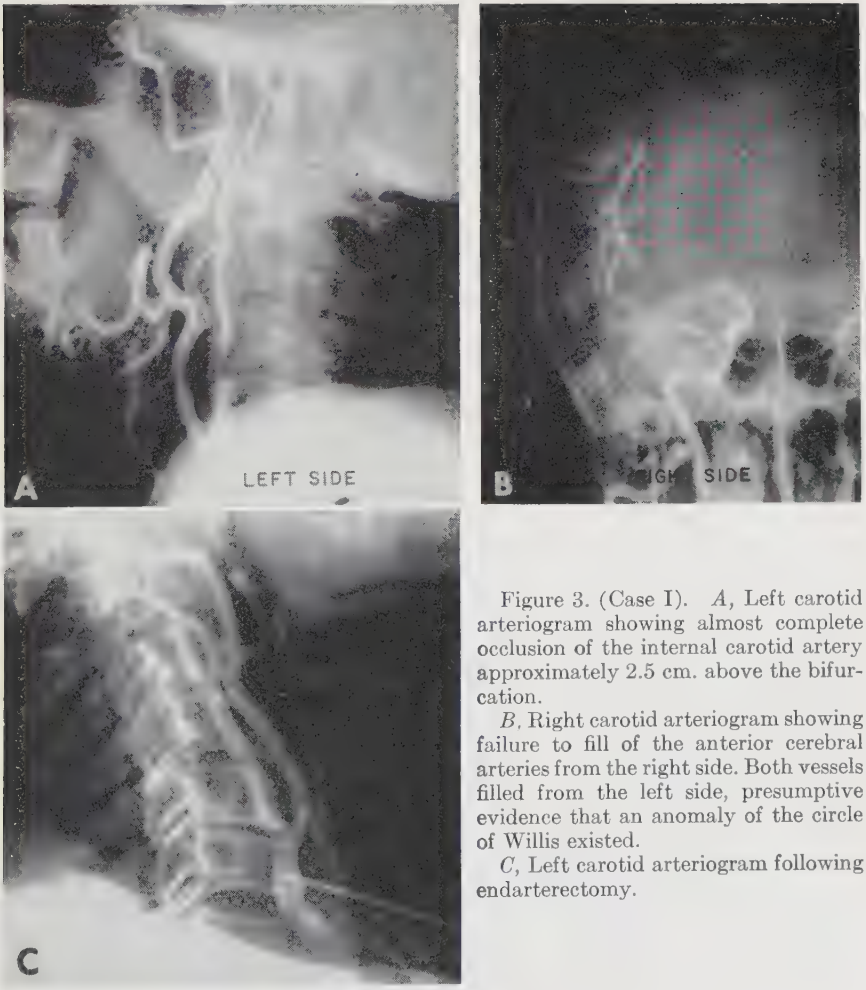

Figure 3. (Case I). *A*, Left carotid arteriogram showing almost complete occlusion of the internal carotid artery approximately 2.5 cm. above the bifurcation.

B, Right carotid arteriogram showing failure to fill of the anterior cerebral arteries from the right side. Both vessels filled from the left side, presumptive evidence that an anomaly of the circle of Willis existed.

C, Left carotid arteriogram following endarterectomy.

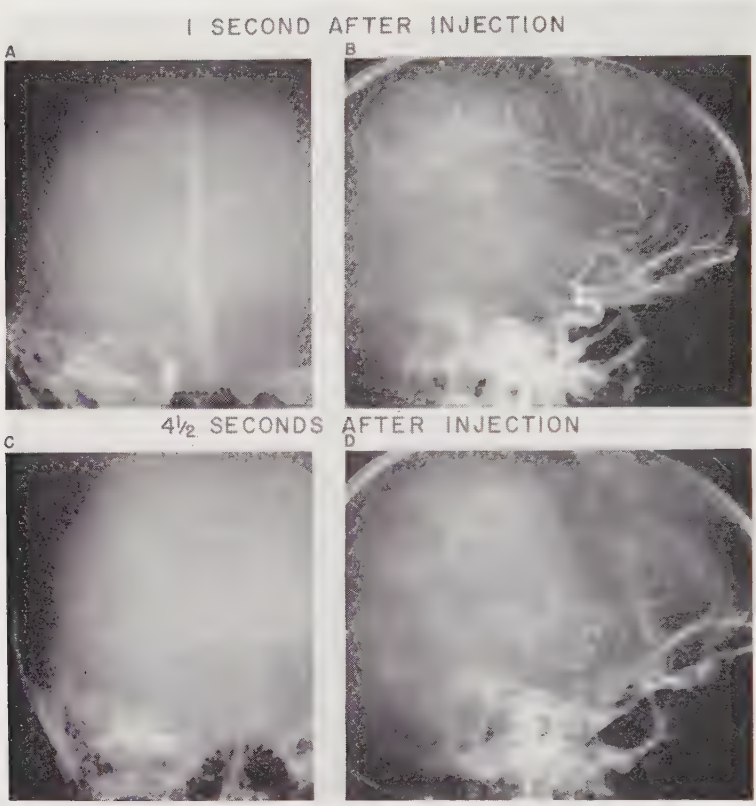

Figure 4. Right carotid arteriogram demonstrating occlusion of the middle cerebral artery one second after injection (*A*, *B*). Four and one-half seconds after injection, fill of the middle cerebral artery is demonstrated beyond the point of occlusion, presumably from pial anastomoses (*C*, *D*).

omy was only two-thirds closed, the occlusion was released with prompt resumption of breathing. The artery was again occluded and the closure completed. The blood loss was replaced. This man's postoperative convalescence was satisfactory and his hemiparesis has continued to improve. Figure 3, *C*, shows his postoperative arteriogram.

The presence and functional significance of pial anastomotic pathways has been documented.[4, 22, 27] In all likelihood their importance lies in establishing collateral circulation between the cerebral arteries following proximal occlusion of one of them, either by thrombosis or embolism (Fig. 4).

A third important source of collateral circulation stems from pathways between various components of the extracranial circulation.[2, 4, 15, 19] Most often this occurs between the internal and external carotid arteries via the ophthalmic artery (Fig. 5) or, occasionally, through the middle meningeal artery. Anastomoses may also take place between the external carotid and

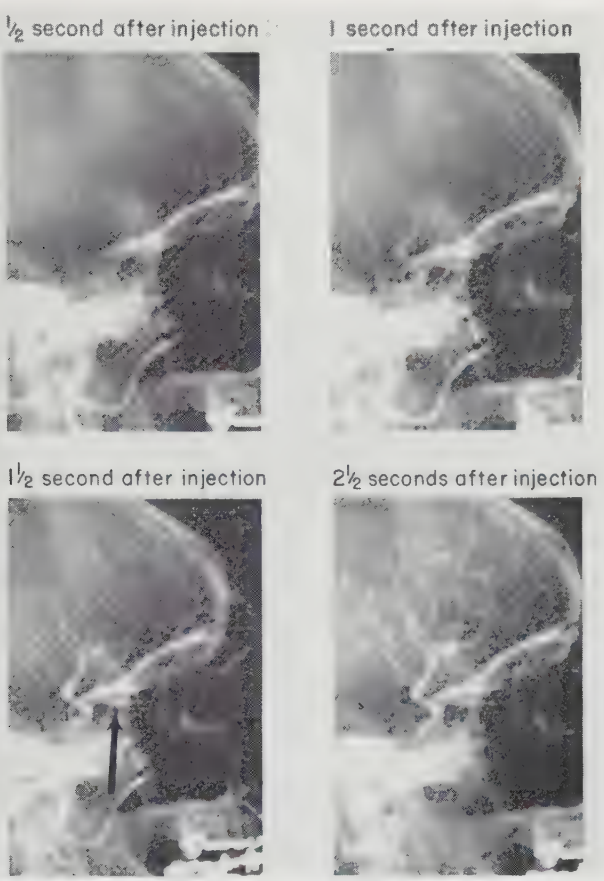

Figure 5. Left carotid arteriogram demonstrating good collateral circulation via the ophthalmic artery in the face of complete left internal carotid artery occlusion.

vertebral arteries and, at times, between the two internal carotid arteries.

This process has been said to take weeks or even months to develop[4, 15] and its demonstration has been presumptive evidence of cervical carotid artery thrombosis of long standing which is probably not directly responsible for recent neurological changes. On the other hand, experience has shown that this is not always true.

Case II. This 66 year old man developed persistent paralysis down the left side 24 hours before admission.

He was found to have, in addition to a left central facial paresis and a left hemiplegia, rather marked blunting of pain sensation down the left side. A right carotid arteriogram showed complete occlusion of the internal carotid artery with good collateral circulation intracranially via the ophthalmic artery (Fig. 6, *A*).

Carotid endarectomy was carried out 24 hours later in spite of the apparent futility of such a procedure. A large plaque was found completely occluding the internal carotid artery at the bifurcation. Much to our surprise when this was re-

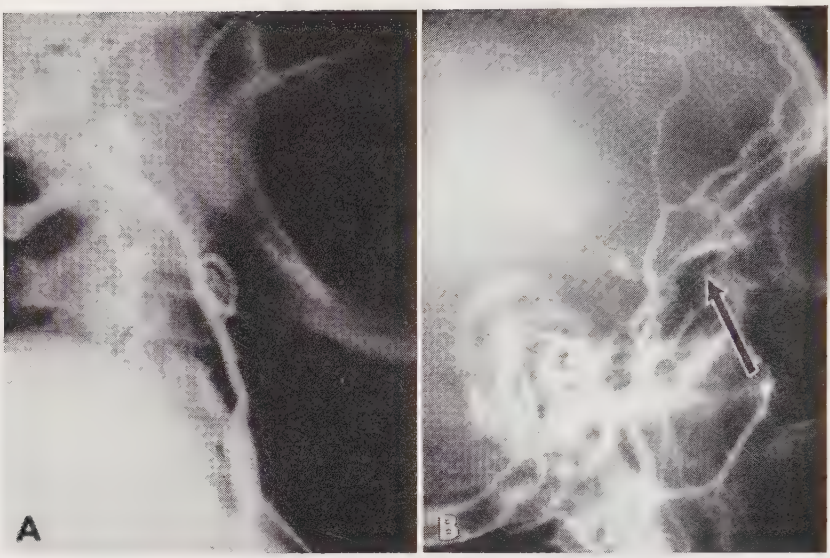

Figure 6 (Case II). *A*, Right carotid arteriogram demonstrating complete occlusion of the internal carotid artery 1 cm. above the bifurcation.

B, Delayed fill of the internal carotid artery via the ophthalmic artery, suggesting falsely that the cervical carotid artery thrombosis was of long standing. (See text.)

moved, good backflow was evident through the internal carotid artery, and postoperative arteriography showed a satisfactory repair. Unfortunately this man has not changed neurologically.

TRANSIENT ISCHEMIC ATTACKS

The occurrence of transient neurological symptoms or "transient ischemic attacks" in patients with carotid insufficiency has aroused much comment. In spite of the fact that the underlying cause is understood, there is no pathological basis for a reasonable explanation of the mechanism producing such episodes since they may represent a physiological change.

Hutchinson and Yates[12] suggested that transient arterial spasm secondary to irritation by repeated hemorrhages into an arteriosclerotic plaque might produce transient neurological symptoms. Present concepts, however, exclude vascular spasm as an exciting cause of transient ischemic attacks,[4, 28] and vascular constriction per se is probably not a factor in their production.

At present the concept of cerebrovascular embolization is emphasized.[4, 8, 9, 18, 21, 23] Arteriosclerotic plaques in any one of the four major arteries in the neck may ulcerate and, themselves, fragment and embolize. In addition, mural thrombi may form at the site of this ulceration and produce emboli. Fisher[8] pointed out that such an embolus might be one mechanism by which an incompletely occluded vessel could produce neuro-

logical signs. He further observed that pathological examination a month later would show no vascular occlusion intracranially for, as a rule, emboli rapidly disintegrate and disappear (Fig. 7). This observation has been mentioned by others.

Systemic factors may also produce this symptom complex. The concept of the "hemodynamic crisis" as set forth by Denny-Brown[4] may explain episodic cerebrovascular insufficiency. In the resting state, significant cervical arterial disease may well be masked by an adequate collateral circulation. However, during periods of stress that may result from hypotension, shock, anemia, hypoglycemia, or even extremes of physical exertion, the collateral circulation may decompensate, producing cerebral ischemia within the distribution of the involved artery. The degree of recovery from such an episode would depend upon the duration of stress. This has been observed both clinically[25] and experimentally.[1]

Case III. A 48 year old, right-handed sailor sustained a head injury and a compound fracture of the left femur in an automobile accident, and was found unconscious at the side of the road some time afterwards. He was brought to the Virginia Mason Hospital in profound shock which was promptly corrected and his

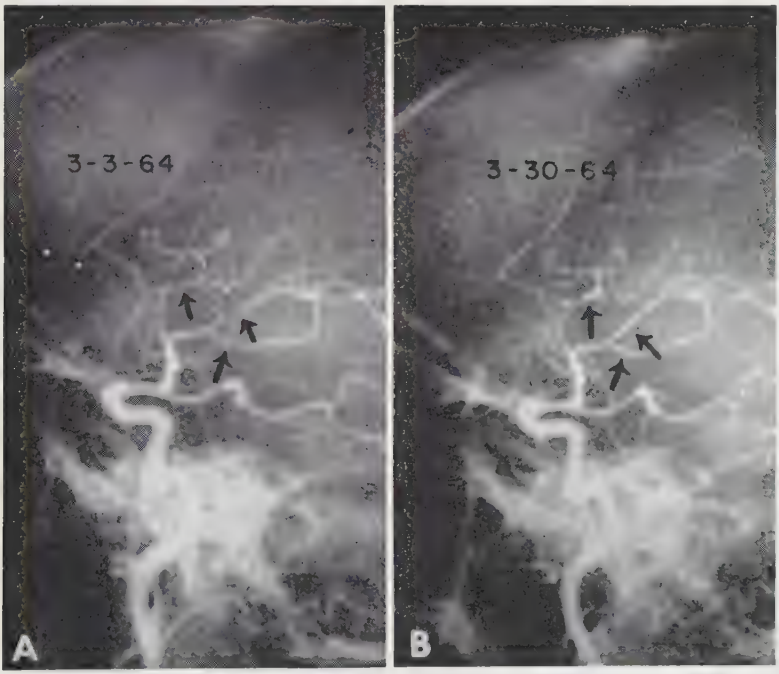

Figure 7. *A*, Left carotid arteriogram in a 43 year old woman who presented with blindness in the left eye, aphasia, and right hemiplegia. The multiple involvement of the middle cerebral artery (arrows) together with the blindness due to retinal artery occlusion, suggested that the process in this case was embolic. No source of embolization could be found.

B, Left carotid arteriogram about one month later showing nearly complete resolution of this process.

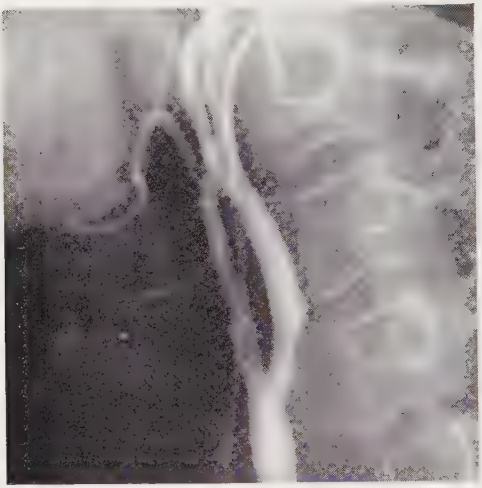

Figure 8 (Case III). Left carotid arteriogram demonstrating stenosis of the internal carotid artery.

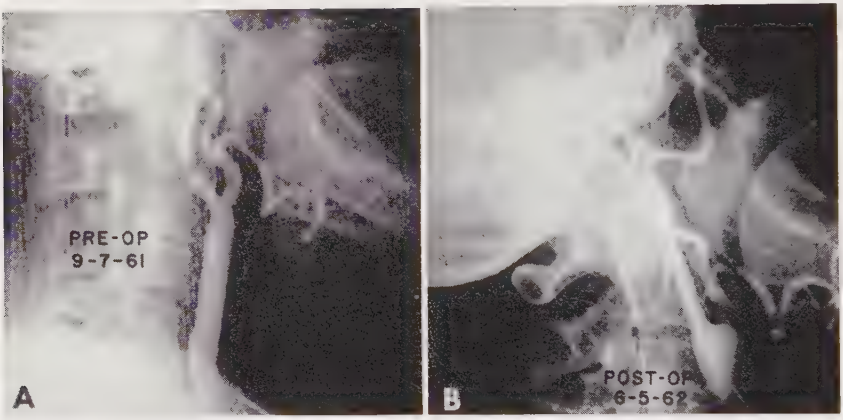

Figure 9. *A*, Right carotid arteriogram demonstrating almost complete occlusion of the internal carotid artery 1 cm. beyond the bifurcation.
B, Right carotid arteriogram 9 months after endarterectomy.

vital signs became stable and normal. He was conscious, but had a pronounced global aphasia. He had a subgaleal hematoma over the left occiput, and a subconjunctival hemorrhage in the left eye. He had a profound right hemiplegia with the arm being involved more than the leg.

In view of this man's relatively intact state of consciousness in the face of profound neurological changes, it seemed more likely that he had sustained a thrombosis of the left internal carotid artery rather than a severe head injury. Arteriography soon afterwards demonstrated a stenosis of the common and internal carotid arteries (Fig. 8), which, in the light of more recent experience, was relatively mild. Unfortunately, he progressed to complete thrombosis following endarterectomy although his neurological picture progressively improved. He was our first case and brought about an abrupt change in surgical technique.

In spite of the slight degree of stenosis encountered in this patient, it

seems reasonable to assume that his total cerebral circulation became inadequate in the face of prolonged shock with its maximum effect occurring within the distribution of the left middle cerebral artery, the area with the least margin of safety.

Figure 9 shows the preoperative and postoperative arteriograms in a 70 year old man who had had a transient blindness of the right eye, precipitated by exhaustion following vigorous garden work.

DIAGNOSIS

The symptoms and signs of carotid artery insufficiency are quite variable since, actually, they reflect the effectiveness of collateral circulation rather than merely the presence of carotid artery occlusive disease. The classical syndrome of ipsilateral blindness with optic atrophy and contralateral hemiplegia has not occurred in any of our patients and in only one case was there amaurosis fugax. Cerebral ischemia, initiated by a discrepancy between cerebral oxygen supply and demand, usually occurs within the distribution of the middle cerebral artery. The resulting symptoms, however, may mimic those from cerebral lesions such as intracranial tumors and subdural hematomas, to say nothing of cerebral vascular disease. A history of transient neurological symptoms suggests carotid artery disease.

Carotid artery palpation and compression carries some risk[20] and has been abandoned by us since, in our hands, it has not been particularly helpful. Most agree that this study is significant only if it is positive. The same applies to retinal artery pressure determinations. Silverstein[26] found a significant diminution in retinal artery pressure in only 65 to 70 per cent of patients with complete carotid occlusion. The presence of carotid bruits has not been particularly helpful in our experience. There have been two instances in which the presence of a carotid bruit has precipitated negative carotid and vertebral arteriograms. In other cases, severe carotid artery stenosis has been demonstrated arteriographically and no bruits have been heard.

A definitive diagnosis can result only from careful, bilateral carotid arteriography. Initially, vertebral-basilar arteriography was performed in this clinic only if it seemed indicated. It is becoming increasingly apparent, however, that all four of the arteries in the neck should be visualized, including their sites of origin.[16]

Case IV. A 54 year old man developed a transient left hemiplegia 4 days after a mastoidectomy. He had had transient signs down the right side 5 years before and in the preceding 3 months or so had had several attacks of unconsciousness.

He was found to be dull mentally and had a residual left hemiparesis. There were bruits over both carotid arteries, louder on the right. The left carotid and left radial pulses were not obtainable. A right carotid arteriogram showed marked stenosis of the internal carotid artery just beyond the bifurcation, along with oc-

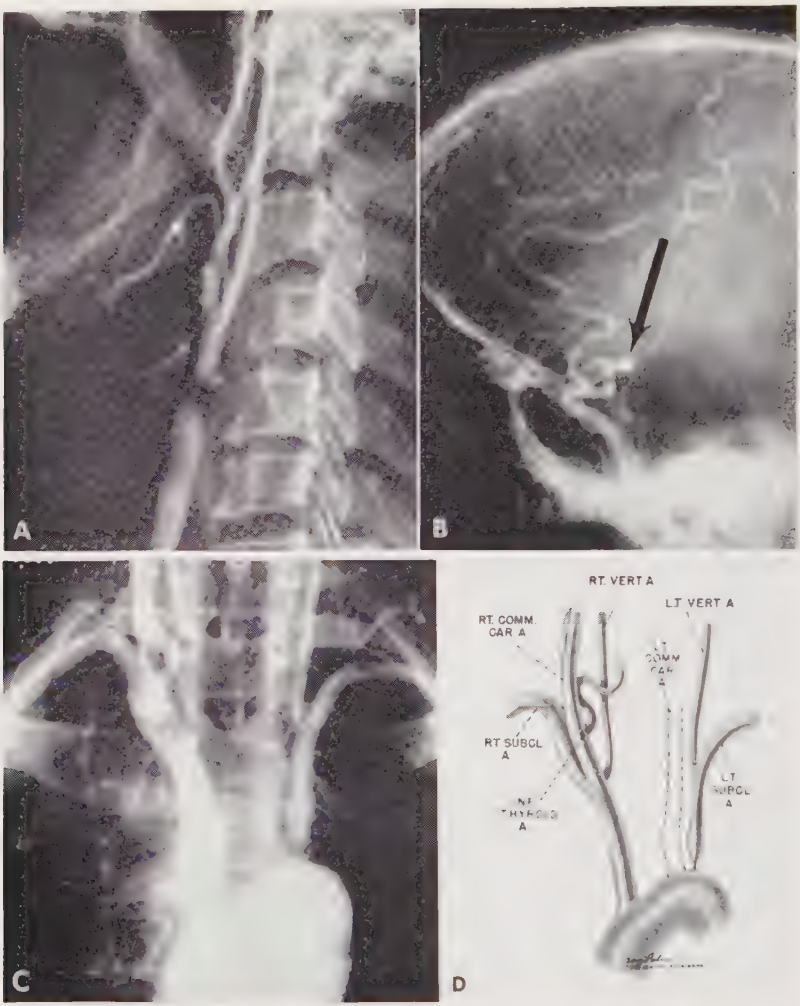

Figure 10 (Case 4). *A*, Right carotid arteriogram demonstrating almost complete occlusion of the internal carotid artery at the bifurcation.

B, Right carotid arteriogram demonstrating thrombosis of the first portion of the middle cerebral artery.

C, *D*, Femoral arteriogram demonstrating marked stenosis of the left subclavian artery and a complete failure of the left common carotid artery to fill.

clusion of the first part of the right middle cerebral artery (Fig. 10, *A*, *B*). A left carotid arteriogram could not be done. Two days later a femoral arteriogram demonstrated marked stenosis of the left subclavian artery and failure to fill of any portion of the left common carotid artery (Fig. 10, *C*, *D*).

At operation, a bypass graft was placed between the aortic arch and the left subclavian artery which reduced the pre-bypass pressure gradient of 60 mm. of mercury between the two vessels to zero. The left carotid complex had become a fibrous cord. A right carotid endarterectomy was done with no change in the pressure gradient of 70 mm. of mercury between the common and internal carotid arteries.

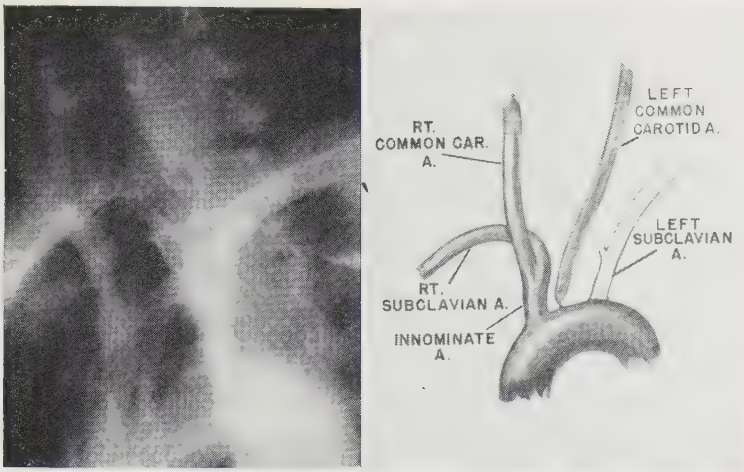

Figure 11. Angiocardiogram showing marked stenosis of the left common carotid artery and complete failure of the left subclavian to fill.

Upon reopening the carotid bifurcation, a small displaced plaque was encountered in the internal carotid artery which was removed. A plastic repair was again carried out and afterwards no pressure gradient was found between the two vessels.

This man has returned to work and has had no recurrence of his symptoms 8 months after operation.

Figure 11 is the angiocardiogram of a 57 year old man who presented one week after developing a transient aphasia and right hemiparesis following exertion. Bilateral carotid arteriograms were negative but angiocardiography was carried out because of diminished left subclavian and radial pulses. This showed complete occlusion of the left subclavian artery along with stenosis of the left common carotid artery at its take-off from the aortic arch. This man has been free of central nervous system symptoms five years following a bypass graft from the ascending arch of the aorta to both the left subclavian and left common carotid arteries.

TREATMENT

The purpose of treatment is threefold:

1. To prevent cerebral infarction from thrombosis or embolism. It will have no effect on an infarction that has already occurred. It has been shown that within 20 seconds of cessation of cerebral blood flow available tissue oxygen is consumed,[4] and that after three minutes at normal temperatures irreversible cerebral ischemia supervenes.[3] "Good results" here can be attributed to the natural course of this process—one of improvement or recovery in approximately 55 per cent.

2. To improve collateral circulation. The effects of progressive cerebral vascular disease may be lessened thereby, and cerebral ischemia from future

atherosclerotic changes in the remaining cervical arteries may be avoided. Arteriographic evidence of external carotid-ophthalmic artery anastomosis does not necessarily signify a favorable prognosis.[16] It suggests inadequacy of other collateral sources and may not be sufficient during periods of stress.

3. To relieve symptoms of transient cerebral ischemia. By improving the total cervical blood supply, the margin of safety is increased and the cerebral effects of hemodynamic crises may be avoided. In a study of 97 patients with carotid artery disease who had as their initial signs rapidly recurring and reversible neurological manifestations, Luessenhop[16] found that 80 per cent had permanent neurological changes at the end of one year.

Experience has shown that if total occlusion of a cervical carotid artery has been present for over 48 hours, endarterectomy is virtually fruitless. After this period of time, the clot organizes and becomes densely adherent to the surrounding intima. Moreover, it propagates superiorly so that its uppermost portion becomes inaccessible.

Surgical treatment is most effective in those patients having intermittent ischemic attacks, without permanent neurological damage and in whom complete occlusion has not taken place. If significant bilateral carotid artery disease is present, bilateral endarterectomy should be considered.

Patients having significant cervical carotid artery disease, but whose symptoms stem from vertebral-basilar insufficiency, may also benefit from carotid endarterectomy.[6] By this means improved basilar arterial supply might result from increased collateral circulation through the posterior communicating arteries. This may be significant since, because of their small caliber, the vertebral arteries have not been amenable to direct surgical attack. Obviously, it is necessary first to demonstrate, arteriographically, an intact basilar artery.

Surgical Technique

In our opinion simple endarterectomy is the treatment of choice. We employ general anesthesia and, using phenylephrine (Neo-Synephrine), induce hypertension during the period of arterial occlusion to maintain an adequate cerebral circulation. In the past, we have used a temporary shunt on patients who could not tolerate carotid occlusion preoperatively. Early in our experience, however, we found this procedure both cumbersome and unnecessary and have abandoned it.

We do not employ hypothermia, having found no need for it even in those patients with complete occlusion of the contralateral carotid artery. Moreover, to be effective, this technique must increase the risk of fibrillation in the face of possible coronary artery disease so frequently encountered in patients of this age.

A Y-type incision is made in the distal common carotid artery to include both the internal and external carotid arteries. The plaque is

removed by gentle dissection, care being taken to preserve the media. Every attempt is made to remove all of the plaque proximally and distally, leaving cuffs of intact intima. In no instance have we found it necessary to employ cuff sutures distally since the intima here is relatively normal and adherent to the media. The incision is reapproximated in such a way that the carotid bifurcation is, if anything, larger than before. The external carotid circulation is preserved because of its importance as collateral blood supply.

Intra-arterial pressure studies are obtained above and below the level of obstruction before and after endarterectomy (Fig. 12). The postendarterectomy pressure determination is especially important in order to be certain that removal of the plaque is complete. This has been emphasized in Case IV.

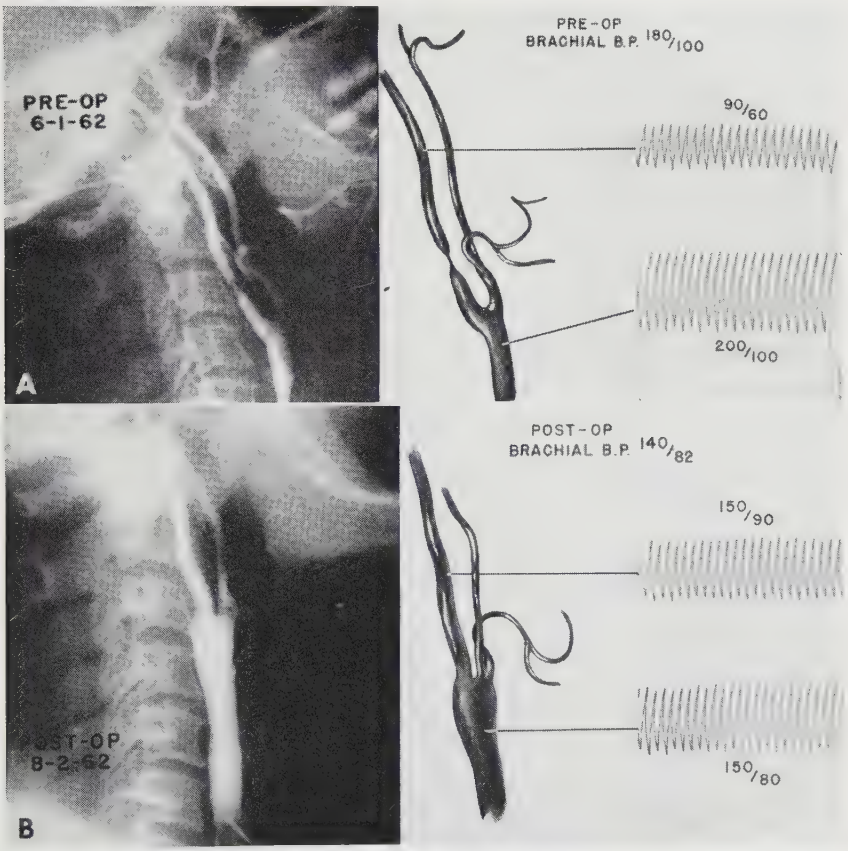

Figure 12. Intra-arterial pressure studies in a 58 year old man with severe stenosis of the left internal carotid artery.

A, Before endarterectomy.

B, After endarterectomy.

SUMMARY AND CONCLUSIONS

The clinical state of patients with carotid artery insufficiency varies considerably since it depends mainly upon two factors: (1) the sum total blood supply from all four extracerebral arteries and (2) the collateral circulation, especially the integrity of the circle of Willis.

Stenosis of one or more cervical arteries may occur without any symptoms, although the margin of safety is lessened. If total occlusion has taken place, there still will be no symptoms in the presence of an intact circle of Willis. If, however, the circle of Willis is defective, symptoms of cerebrovascular insufficiency will occur which, depending upon the magnitude of this defect, may be severe and long lasting.

A hemodynamic crisis, occurring in the presence of stenosis of one or more of the cervical arteries, may act as an exciting cause to produce an ischemic attack, the severity of which will depend upon: (1) the duration of the exciting cause, (2) the competency of the circle of Willis, (3) the total blood supply available from all four extracerebral arteries, (4) the degree of cerebrovascular disease.

Embolization from a plaque in any one of the cervical arteries may also produce an ischemic attack, the severity of which will depend upon the size of the embolus and the effective cerebral collateral circulation.

Considering these factors, clinical assessment may be suggested but never definitive. Arteriography is the only means whereby an accurate appraisal can be assured.

The main purpose of treatment is to prevent cerebral infarction. Untreated, roughly 80 per cent of patients with transient neurological symptoms in the face of carotid artery occlusion will progress to cerebral infarction within one year. Treatment directed to reversing a stroke that has already occurred is obviously futile since, without oxygen, the brain can survive only three minutes. It may, however, be effectively applied to prevent another one.

One-fourth of the patients with symptoms of cerebrovascular insufficiency may have significant extracerebral vascular disease. Many of these patients may derive benefit from carotid endarterectomy, especially if total carotid occlusion has not occurred.

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Surgical Anatomy of the Lungs

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Precise surgical treatment of pulmonary disease requires an accurate understanding of the bronchovascular anatomy of the lungs.

Early studies in this field were by Aeby (1880) and Ewart (1889). A more accurate description of the bronchial tree and the introduction of the term, bronchopulmonary segment, by Kramer and Glass (1932) preceded the successful segmental resection of the lingula for bronchiectasis by Churchill and Belsey in 1939. Foster-Carter (1942) introduced the term, segmental bronchi. Then in 1943 the bronchoscopist Jackson and the anatomist Huber described the now standard terminology of the pulmonary segments. Brock (1954) in his classical studies of lung abscesses added an outstanding contribution, making possible the accurate localization of lesions in the pulmonary segments. Boyden (1955) and Bloomer et al. (1960) accomplished the monumental task of correlating the bronchial and vascular patterns of the segments and recording the variations in these arrangements.

This article will present a concise account of the structure of the lungs as related to thoracic surgery. For a more detailed discussion the aforementioned original works should be consulted.

The Lobes and Fissures

The slightly larger right lung with its upper, middle and lower lobes is anatomically similar to the left lung with its upper and lower lobes when one considers the middle lobe as being represented by the lingula on the left side.

The oblique fissure separates the lower lobe from the upper lobe on the left, and from the upper and middle lobes on the right. It begins posteriorly at the level of the fifth rib or interspace (slightly higher on the

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left) and then follows approximately the course of the sixth rib. The horizontal fissure separating the middle and right lower lobes starts from the oblique fissure in the midaxillary line at the level of the sixth rib and runs transversely to the fourth costal cartilage. An anterolateral thoracotomy in the fourth intercostal space thus gives excellent exposure to the middle lobe and hilum for operations on the middle and lower lobes. This submammary incision is cosmetically more desirable, and with less division of the large back muscles there is less disability than with the standard posterolateral approach.

Hilum

The principal structures at the pulmonary hilum (Fig. 1, *A*, *B*) are the main bronchus, the pulmonary artery, and superior and inferior pulmonary veins. The pulmonary artery occupies a different position on the two sides. The left pulmonary artery, a direct continuation of the main pulmonary artery, passes around superior and posterior to the upper lobe bronchus and is the highest structure in the hilum, whereas the right pulmonary artery, arising at right angles from the main trunk, passes posterior to the aorta and superior vena cava to enter the lung anterior and inferior to the bronchus.

The superior pulmonary veins are anterior and inferior to the pulmonary arteries, and the inferior pulmonary veins are still further inferior and on a more posterior plane.

The pleura, which covers the anterior and posterior aspects of the hilum, joins below to form the pulmonary ligament which extends between the medial surface of the lower lobe and the pericardium. Pulmonary resection involving the lower lobe requires division of this ligament. A landmark for the upper limits of this dissection is a lymph node situated just below the inferior pulmonary vein.

On the right the azygos vein ascends the mediastinum posterolateral to the esophagus and arches over the hilum to enter the superior vena cava. The phrenic nerve descends on the superior vena cava and then on the pericardium approximately 1 cm. anterior to the hilum.

On the left the aorta arches over and then descends posterior to the hilum. The phrenic nerve takes a perpendicular course beneath the mediastinal pleura, passing about 1.5 cm. anterior to the hilum. The vagus, after giving off the recurrent laryngeal nerve at the ligamentum arteriosum, passes behind the hilum to descend on the esophagus.

The Pulmonary Segments

The lobes of the lungs are divided into segments (Fig. 2), each with its own bronchus, pulmonary artery and vein. The respective bronchi and pulmonary arteries are in close association, centrally placed in the segment or subsegment, whereas the pulmonary veins are peripheral and intersegmental (Fig. 3). The terminology of the bronchovascular segments has

been standardized with the adoption of the Jackson and Huber nomenclature for ten segments on the right and eight on the left. A common stem bronchus supplies the apical-posterior segment of the left upper lobe and, likewise, the anteromedial basal segment of the left lower lobe, thus explaining the different number of segments on the two sides.

The boundaries of the segments cannot ordinarily be seen, but occasionally there are clefts or fissures separating a segment from the parent lobe. This anomaly affects not infrequently the superior segment, the medial basal segment, and the lingula.

Tracheobronchial Tree

In the adult the trachea is approximately 11 cm. in length, bifurcating at the level of the seventh thoracic vertebra as confirmed by roentgenological studies in the living (Brock).

The right main bronchus (Fig. 4, *A*) is shorter, wider and more vertical than the left. The right upper lobe bronchus, arising within 2 cm. of the carina, divides into bronchi to the apical, anterior and posterior segments. The bronchus between the upper and middle lobe branches is termed the intermediate bronchus (pars interlobaris). At its termination it gives origin on its anterior aspect to the middle lobe bronchus supplying medial and lateral segments of this lobe, and opposite on its posterior aspect to the superior segmental bronchus of the right lower lobe. Then a short common basal bronchus gives rise successively to medial, anterior, lateral and posterior basal bronchi.

When performing a lower lobectomy, it is frequently advisable to divide and suture the common basal bronchus and the superior segmental

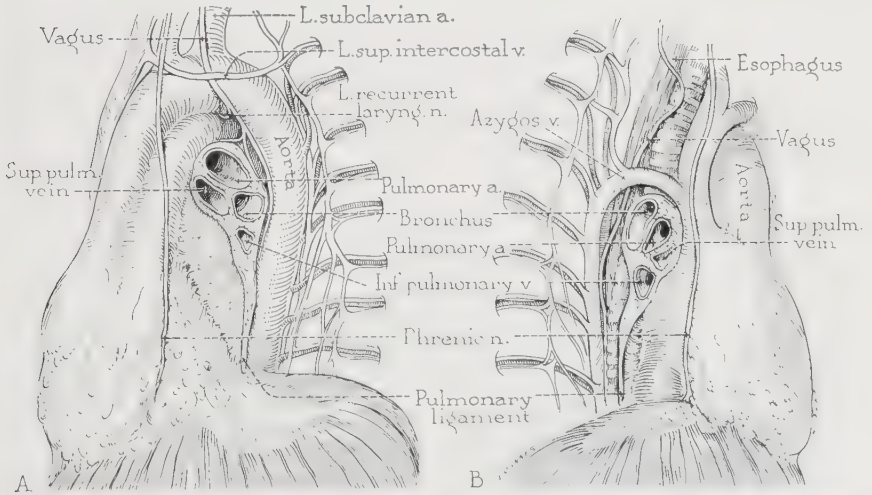

Figure 1. The mediastinum as viewed from (*A*) left and (*B*) right sides, showing the relations of the bronchi and vessels at the hilum.

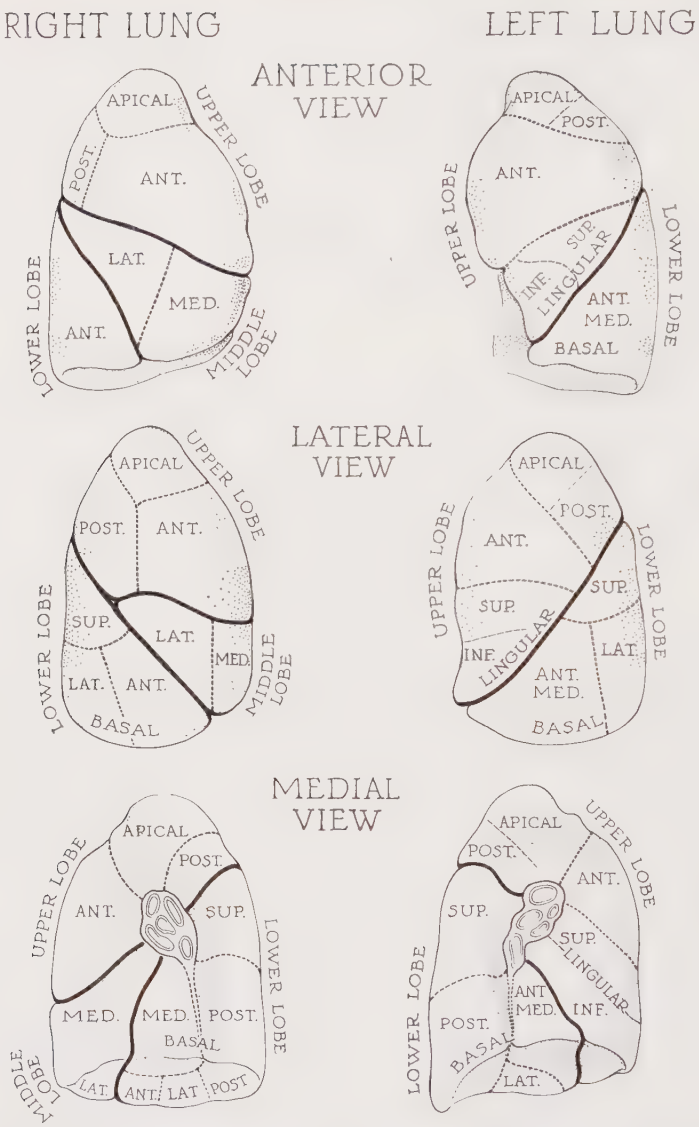

Figure 2. The pulmonary segments of right and left lungs as seen from anterior, lateral and medial aspects.

bronchus separately because of the proximity of origins of the superior segment and middle lobe bronchi.

The left main bronchus is over twice as long as the right (Fig. 4, *A*), the left upper lobe bronchus arising 4 to 6 cm. from the carina. The upper lobe bronchus has a superior and inferior division, the superior dividing into the anterior and apical-posterior segmental bronchi. The inferior division divides to supply superior and inferior segments of the lingula

which are analogous to the lateral and medial segments of the right middle lobe.

The left lower lobe bronchus is very short, giving off the superior segmental bronchus less than 1 cm. from the upper lobe orifice, and again it is advisable to divide the superior segmental and common basal bronchi separately in doing a left lower lobectomy.

The bronchi of the left lower lobe are similar to the right except that the medial and anterior segmental bronchi arise from a common stem.

The bronchoscopic view of the tracheobronchial tree, showing the orifices of the main, lobar and segmental bronchi, is seen in Figure 4, *B*.

Bronchial Variants

Rarely, variations occur in the origin of bronchi, and these variations may be sufficiently marked to justify description as displaced or supernumerary bronchi (Brock).

Displaced Bronchi. A bronchial subdivision may slip up or down in relation to its usual site of origin. The most common is an upward displacement of part or all of the apical bronchus of the right upper lobe. Other segments of the right upper lobe may be similarly displaced.

Supernumerary Bronchi. No bronchus should be labeled supernumerary unless there is clear evidence that all the normal bronchi are present as well as the abnormal bronchus. The most common supernumerary bronchus arises from the trachea or right main stem bronchus to an additional segment in the right upper lobe.

Pulmonary Arteries

The pulmonary arteries are more variable than the bronchi, but, in general, they divide to closely follow the segmental and subsegmental bronchi, usually on their superior or lateral surfaces, and carry the same names.

The right pulmonary artery (Fig. 5, Anterior), just lateral to the superior vena cava, gives rise to the truncus anterior, supplying the apical

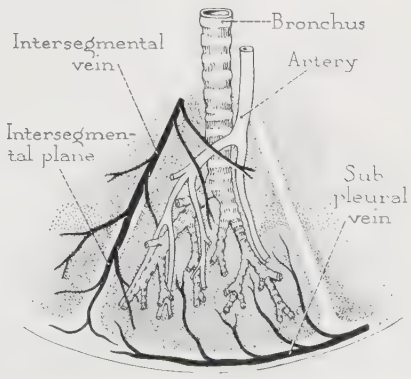

Figure 3. A diagrammatic representation of the bronchovascular supply to a pulmonary segment.

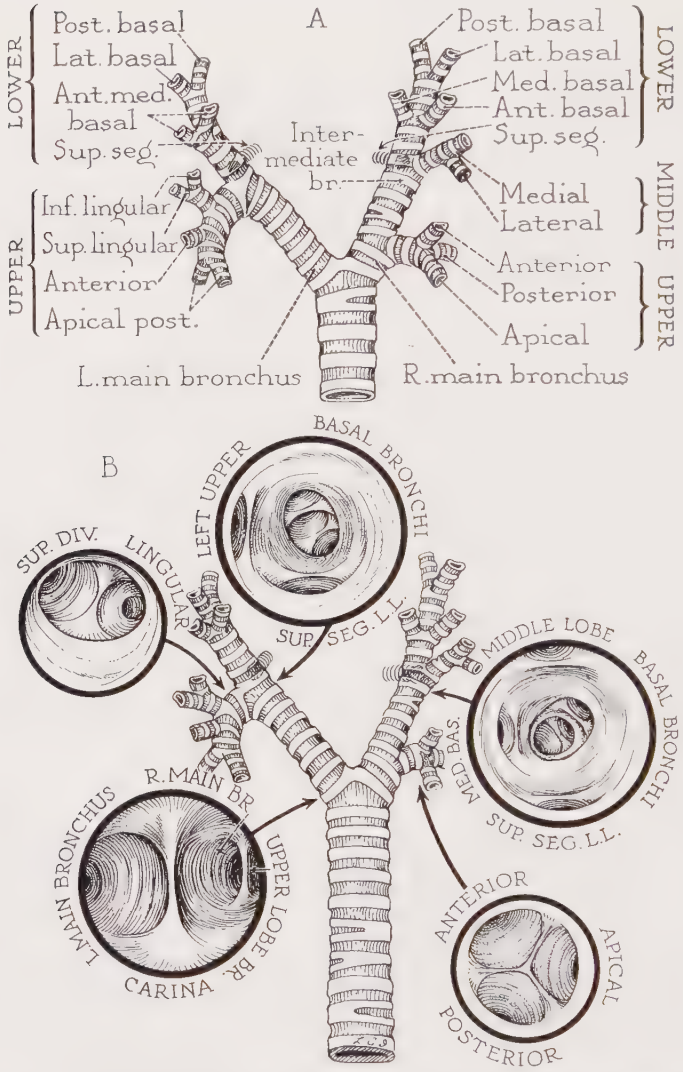

Figure 4. The tracheobronchial tree showing the origins of the main, lobar and segmental bronchi: *A*, External aspect, and *B*, bronchoscopic view.

and anterior segments of the upper lobe. The pulmonary artery then descends lateral to the intermediate bronchus. From its interlobar portion (pars interlobaris) arises a branch or branches to the upper lobe (posterior ascending artery) (Fig. 5, Fissure). The middle lobe receives one or two arteries and then branches are supplied to the superior segment of the lower lobe, and medial, anterior, lateral and posterior basal segments.

The left pulmonary artery arches over the upper lobe bronchus, making a sharp turn to enter the floor of the interlobar fissure, where it is lateral

to the lower lobe bronchus. The first arterial branch to arise from the pulmonary artery (Fig. 6, Anterior) is commonly distributed to the anterior segment and/or lingula. Next, branches to the apical and posterior segments arise as separate branches or as a single trunk. The first branch to the lower lobe, the superior segmental artery, arises from the lateral aspect of the artery high in the fissure (Fig. 6, Fissure) usually a little higher than the final branches to the upper lobe, namely, those to the lingula. The artery terminates by supplying the three basal segments. In a left lower lobectomy, it is usually necessary to ligate the superior segmental

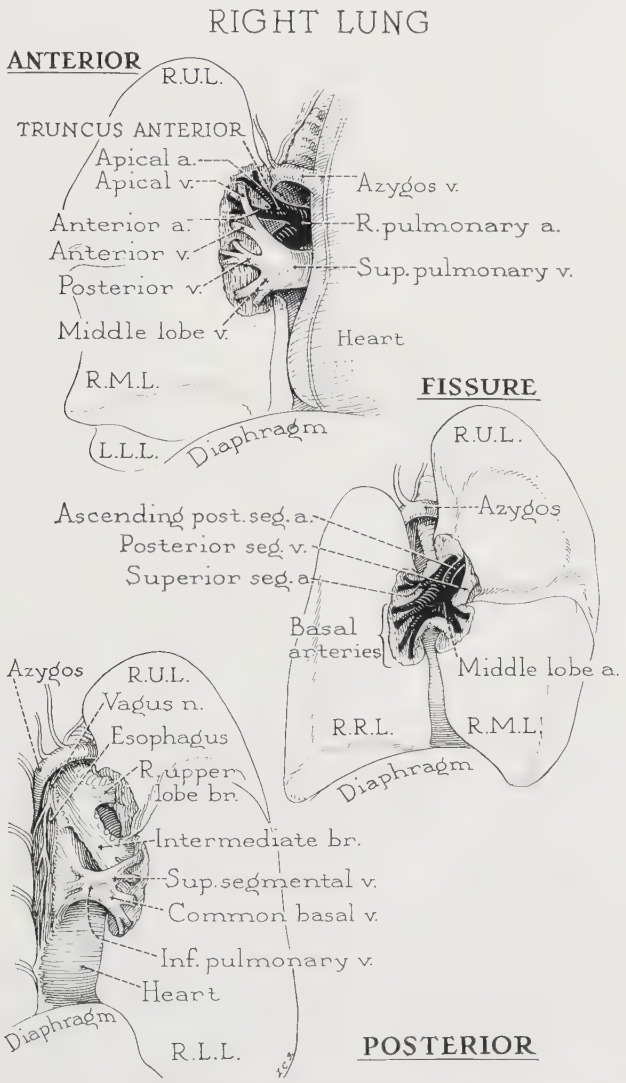

Figure 5. The vascular supply to the right lung: anterior, fissure and posterior views.

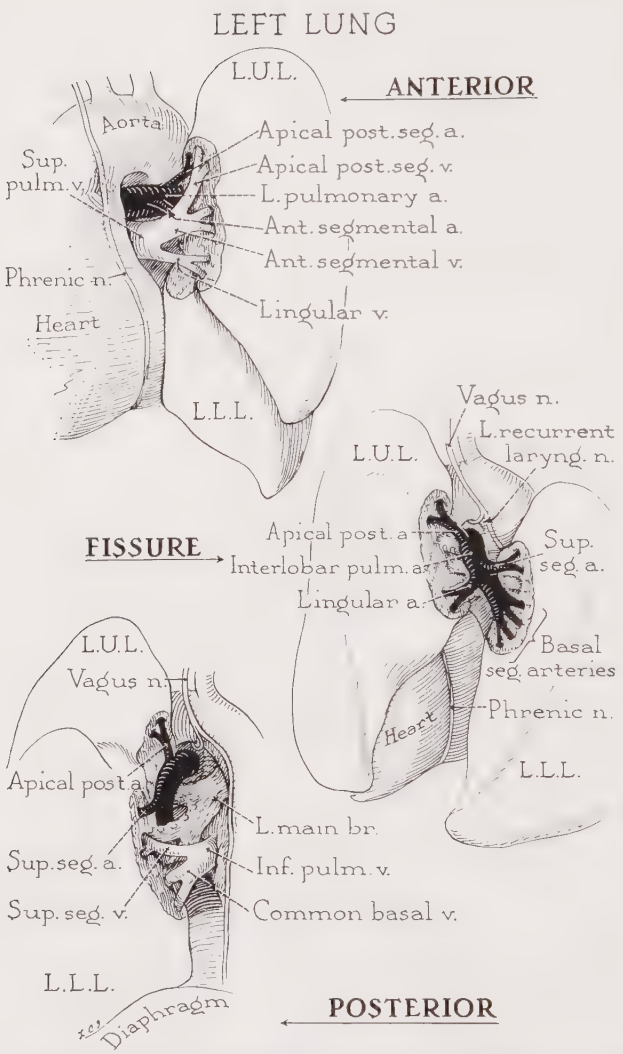

Figure 6. The vascular supply to the left lung: anterior, fissure and posterior views.

artery separately from the basal arterial trunk so that the blood supply to the lingula is not compromised.

Pulmonary Veins

There is considerable variation in the pulmonary venous drainage of the lungs. In the periphery, the branches of the pulmonary veins are intersegmental (Fig. 3) and drain the bordering segments. When segmental resection is performed, these veins should be preserved to avoid congestion of adjacent segments.

With few exceptions, there is a superior and inferior vein bilaterally. The superior vein drains the upper and middle lobes on the right, and the upper lobe, including the lingula, on the left. The inferior pulmonary veins drain the respective lower lobes.

The right upper lobe has three veins, apical, anterior and posterior, draining into the superior pulmonary vein (Fig. 5, Anterior). The posterior vein has a central and interlobar division. It crosses the pulmonary artery in the interlobar fissure (Fig. 5, Fissure) and must be avoided when dissecting for the posterior ascending artery to the upper lobe. The middle lobe vein or veins also drain into the superior pulmonary vein. The right lower lobe is drained by the inferior pulmonary vein, formed by the junction of the superior segmental and common basal veins (Fig. 5, Posterior).

The left superior pulmonary vein drains the left upper lobe through three main trunks, apical-posterior, anterior and lingular (Fig. 6, Anterior). The apical vein overlies the first branches of the left pulmonary artery and is in close proximity to their origins. During dissection of the first branches of the left pulmonary artery, care must be taken to avoid injury to the apical vein. As on the right, the inferior pulmonary vein is formed by the junction of the superior segmental vein with a common basal vein.

A lymph node lies immediately below each inferior pulmonary vein at the uppermost extent of the pulmonary ligament.

The Bronchial Arteries

The bronchial arteries arise most commonly from the aorta, just distal to the arch, and vary in number from one to three to each lung. Careful ligation of these vessels during pulmonary resection is essential; otherwise, hemorrhage from these arteries may require a second thoracotomy. The bronchial arteries may be enlarged in certain types of cyanotic congenital heart disease and in chronic pulmonary disease.

Intrapericardial Ligation of the Pulmonary Vessels

Intrapericardial ligation of a vessel may be required when tumor encroaches upon the pericardium, inflammatory adhesions prevent hilar dissection, or vessels are accidentally torn during surgery. The right and left pulmonary arteries and the four pulmonary veins can be seen intrapericardially as protrusions posteriorly in the serous pericardium (Fig. 7). By an incision of the posterior pericardium at the superior and inferior margins of the vessel, its posterior surface may be dissected and a ligature passed around it. On the right the incision in the pericardium is made anterior to the phrenic nerve (Fig. 7, *A*). This gives access to the right pulmonary artery between the aorta and superior vena cava. The right pulmonary veins may be isolated where they enter the left atrium. On the

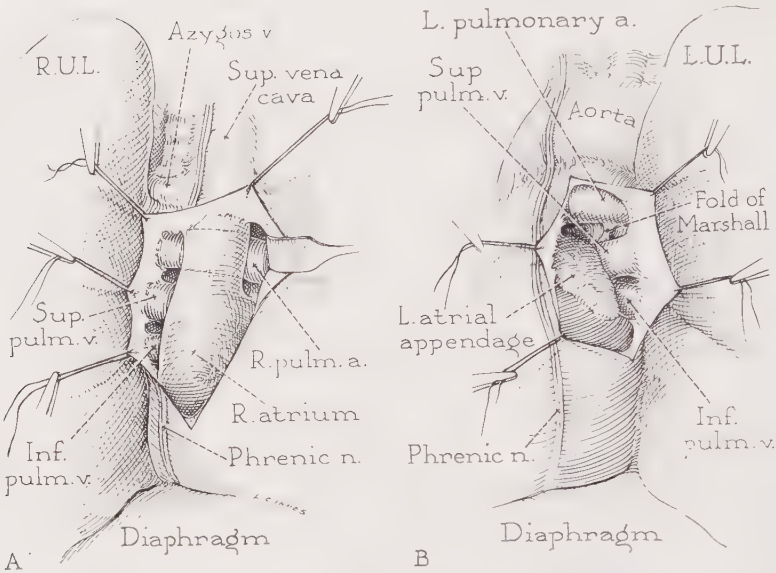

Figure 7. The intrapericardial relationships of the pulmonary arteries and veins. In pulmonary surgery ligation of these vessels within the pericardium may be necessary.

left the pulmonary artery in its short intrapericardial course may be ligated at the fold of Marshall (Fig. 7, *B*).

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Esophageal Sphincter and Reflux

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Gastroesophageal reflux with its attendant complication of peptic esophagitis is an extremely common clinical problem which at times is severely incapacitating and life threatening. An understanding of the esophagogastric junctional area which normally contains the sphincteric mechanism for preventing reflux has proved to be extremely elusive. Of particular importance is the problem of reflux esophagitis in association with sliding hiatal hernia. Although much progress has been made, surgical measures remain unsatisfactory in many respects—so much so that the gastroenterologist is often hesitant to ask for surgical intervention until the most exhaustive and stringent medical measures have been tried. This attitude in large measure is the result of a relatively high incidence of surgical failures, particularly when these patients are followed on a long-term basis. Nonetheless, real progress has been made and it is the purpose of this brief presentation to attempt to consolidate these advances into a meaningful concept.

GROSS AND MICROSCOPIC ANATOMY

It is not the purpose of this article to review and examine the various theories of the cardiac sphincteric mechanism and the reader is referred elsewhere for an analysis of this material (Mann, Greenwood and Ellis). It is the author's opinion that the evidence clearly indicates that there is an intrinsic esophageal sphincter at the lower end of the esophagus which is primarily responsible for preventing gastric reflux. The concept outlined in this article is based on this premise and the following anatomic and histologic observations.

Normally, the tubular esophagus extends for a variable distance

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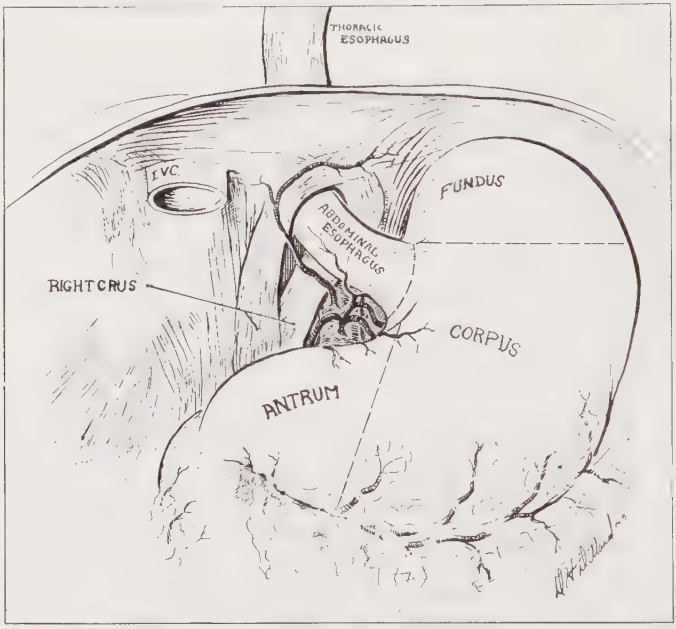

Figure 1. Diagrammatic representation of the "normal" anatomy of the esophagogastric junction. The peritoneum and endoabdominal fascia have been removed, revealing a short segment of intra-abdominal esophagus. The hiatus in this specimen is made up solely from the right crus of the diaphragm.

(approximately 1 to 4 cm.) below the level of the diaphragm before abruptly enlarging to form the stomach (see Fig. 1). Although an acute angle of esophageal entry is formed by the left lateral wall of the esophagus and the ascending domelike fundus, these two structures frequently are not in direct apposition to one another and hence no flap valve mechanism is present. Posteriorly, the esophagus is bounded by the crura of the esophageal hiatus and the aorta; to the right and anteriorly by the caudate and left lobes of the liver; to the left posterolaterally by the inferior surface of the diaphragm, and to the left anterolaterally by the liver. The lower esophagus is anchored to the diaphragm as it passes through the diaphragmatic esophageal hiatus by the phrenoesophageal membrane. This membrane is comprised of a number of components including, in portions of its circumference, the pleura, the supradiaphragmatic fascia, the infradiaphragmatic or endoabdominal fascia, and the peritoneum. The most substantial components of this membrane are contributed by fibers from the endoabdominal fascia. Some of these fibers extend cephalad forming the ascending leaf of the phrenoesophageal membrane, and others extend distally toward the stomach to form the descending leaf of the membrane (see Fig. 2). The leaves of the phrenoesophageal membrane bracket that portion of the lower end of the esophagus which contains the intrinsic sphincter and is called the vestibule by radiologists (Zaino, Poppel et al.).

This localization is based primarily on physiologic data although there is some anatomic evidence (Byrnes and Pisko-Dubienski) that the circular muscle layer in this zone is thickened and somewhat different in character histologically (see also Fig. 3).

The squamous epithelial lining membrane of the esophagus abruptly changes to a columnar glandular type of epithelium within the confines of this membrane. In the dog, this line was found consistently close to the proximal insertion point of the ascending leaf of the phrenoesophageal membrane, whereas in the human specimens examined, this zone of mucosal transition tended to be situated more closely to the insertions of the descending leaf of the phrenoesophageal membrane. This has also been noted by Zaino et al. In both the animal and human specimens, the character of the epithelium immediately adjacent to the squamous epithelium was different from that within the stomach proper. There tended to be shallower crypts and less branching of the glandular structures and there were few to no parietal cells. This corresponds to the zone referred to by Hayward as the zone of junctional epithelium. Teleologically, it seems

Figure 2. Photograph of a longitudinal section of the esophagogastric junctional area of a dog with attached phrenoesophageal membrane and narrow rim (vertical arrow) of the muscular crural edge of the diaphragmatic esophageal hiatus. The specimen was removed intact, fixed in toto and then sectioned longitudinally with the membrane stretched to bring out the insertions and pleural and peritoneal relationship. No dissection of the area to accentuate these structures has been done.

a, The pleura reflecting off the superior surface of the diaphragm.

b, The fascial layer of the ascending leaf of the phrenoesophageal membrane which inserts just cephalad to the squamocolumnar mucosal junction shown at *e*.

c, The descending fascial leaf of the membrane covered by peritoneum shown at *d*. The thickness of the circular muscle of the sphincteric area is seen between the short arrows.

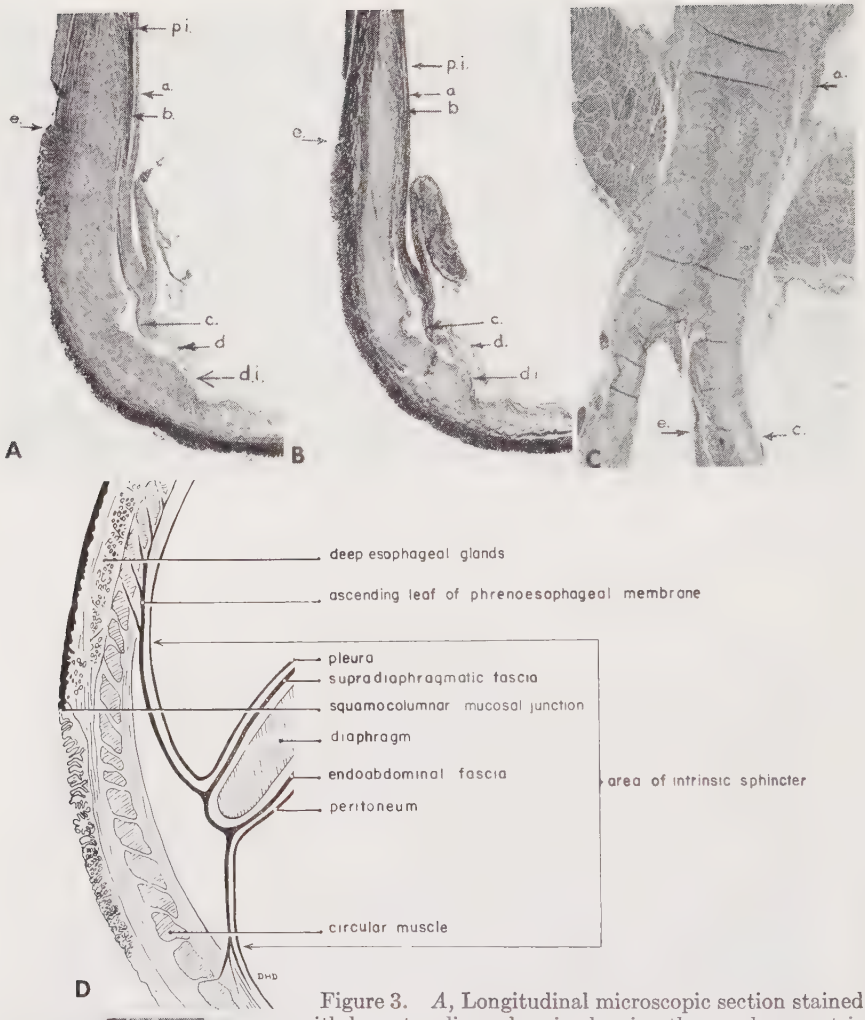

Figure 3. *A*, Longitudinal microscopic section stained with hematoxylin and eosin showing the esophagogastric junction of a dog fixed in the same manner as the specimen in Figure 2.

B, Same specimen as *A*, stained with Verhoeff's and Van Gieson's connective tissue stains. Arrow at *p.i.* shows insertion of ascending leaf of phrenoesophageal membrane with fibers penetrating into the muscular coats of the esophagus. *a* and *b* in sections *A* and *B* show the same layers seen in the gross specimen (*a* and *b* of Figure 2). Note that the squamocolumnar junction (*e*) is below the proximal insertion point of the membrane, but above the point of insertion (*d.i.*) of the descending leaf of the membrane. Note that in this specimen the circular muscle between the insertion points of the membrane is thickened. This corresponds to the area of the physiological intrinsic sphincter and was a frequent finding (see also section *E* from another animal).

C, Longitudinal section through esophagogastric junc-

(*Continued on opposite page*)

reasonable that such a buffering zone of nonacid secretory, but acid peptic resistant, mucosa should be interposed between the extremely sensitive squamous epithelium and the secretory gastric epithelium. Furthermore, it appears convenient that this also corresponds to the zone of the intrinsic sphincter since, ideally, compression and closure of the esophageal lumen at this point ensures an adequate separation of the sensitive squamous epithelium from the acid peptic juices.

Histological section (Fig. 3) reveals that the connective tissue fibers of the phrenoesophageal membrane penetrate into the walls of the esophagus, mixing with the connective tissue of the muscle layers and penetrating into and beyond the circular muscle to the submucosal layer. Examination both radiologically and grossly of specimens in situ reveals that there is a fair degree of mobility of the vestibular area relative to the edge of the esophageal hiatus. Consequently, depending upon the various situations of intra-abdominal and intrathoracic pressure and movements of the diaphragm, this area may shift slightly cephalad or caudad in relation to the hiatus, but the lowermost zone still remains within the abdominal compartment. Furthermore, the fixation or insertion points of the ascending and descending limbs of the phrenoesophageal membrane (although they fan out over a longitudinal zone and do not reach a pinpoint insertion) are relatively fixed and set at their point of junction with the esophageal wall. The stress of their pull is distributed over a wider zone because of intermingling with the connective tissue fibers of the esophagus proper.

Experimental and clinical data currently indicates that the only functionally significant innervation of the sphincteric area of the esophagus is via the vagus nerve. Standard vagotomy at the level of the hiatus experimentally and clinically exerts little effect upon intrinsic sphincteric function. High bilateral vagotomy apparently does exert some influence. The exact role of the vagus nerves, however, remains to be elucidated. The muscular crura of the esophageal hiatus are variable. Eleven different types have been described by Listerud and Harkins. Their findings on a series of autopsy specimens suggested that the incidence of hiatal hernia was not related to the origin of the crus or crura which form the hiatus.

MECHANICAL ANATOMY AND PHYSIOLOGY

Experimental work in dogs shows that following incision of the phrenoesophageal membrane the intrinsic lower esophageal sphincter may function

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tion of human newborn. Ascending leaf (*a*) and descending leaf (*c*) of phrenoesophageal membrane are shown. Note that in this instance the squamocolumnar junction (*e*) is lower than in the dog specimens, but still within the confines of the membrane insertions.

D, Diagrammatic representation of specimens *A*, *B*, *C* and *E*. The designation of the sphincteric area is based on the physiological evidence; however, anatomic evidence is present in many specimens.

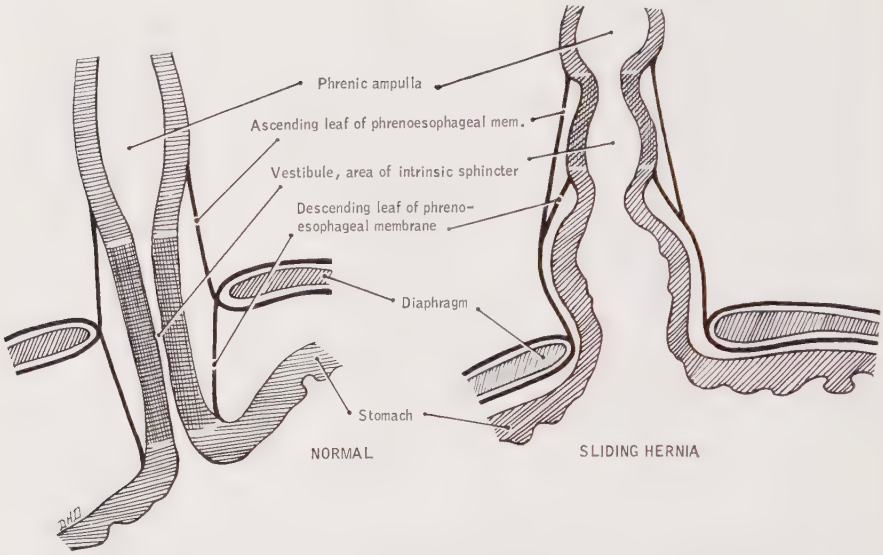

Figure 4. Diagrammatic representation of the anatomy of the esophagogastric junction and relationship of the sphincteric area. In the normal, the intrinsic sphincter is protected from distracting mechanical influences by the phrenoesophageal membrane. In this diagram, the cross-shaded area represents the location of the intrinsic sphincter according to physiological data.

adequately to prevent reflux if the esophagogastric junctional area is displaced above the normal diaphragmatic esophageal hiatus. Furthermore, this sphincter functions satisfactorily in the absence of an acute angle of esophageal entry. Clinical analogy can be found in that many patients with sliding esophageal hiatal hernia do not have sphincteric incompetence. On the other hand, many patients with sliding esophageal hiatal hernia do have reflux and may develop severe peptic esophagitis with a hernia of equivalent size and gross configuration to the above patient without symptoms.

Why should this paradoxical situation exist? Examination of the phrenoesophageal membrane sheds some light on the question. Normally, the sphincteric area is encased in a tube of phrenoesophageal membrane which terminates at the insertion points of the ascending and descending leaves of this membrane (see Fig. 3). Although some connective tissue fibers do traverse the space between the confines of the ligament and the wall of the esophagus, most of the effective restraining influences are at either extremity of this membranous tube. The mechanical arrangement depicted in Figures 4 and 5 demonstrates the action of the phrenoesophageal membrane in resisting herniation of the lower esophagus without interfering with the function of the intrinsic sphincter. The downward pull of the stomach is similarly restrained by the membrane, also without interfering with sphincteric function. Furthermore, enlargement of the hiatus

or lateral distraction of the membrane exerts a relatively small influence on the sphincter. In the presence of sliding esophageal hiatal hernia, however, it is obvious that the fibers from the phrenoesophageal membrane must either stretch or disrupt in order to allow herniation to occur. If this process occurs in such a manner that the effective points of insertion of the membrane are not changed, then it is possible for sliding hiatal hernia to occur and sphincteric function to remain intact (see Fig. 5, *B*). That is to say, it remains intact in the sense that it still constitutes a barrier against reflux. The sphincter, however, operates at some disadvantage since the benefit of the positive pressure reinforcement of intra-abdominal pressure against the lower abdominal esophagus is lost.

In other situations wherein the sliding hiatal hernia has resulted in a disturbance of the normal pattern of the phrenoesophageal membrane so that the effective point of insertion of this membrane has shifted to an area impinging on that of the intrinsic sphincter (Fig. 5, *C*), the sphincter must combat not only the pressure created by intragastric forces, but also the action of the membrane itself which, in restraining the esophagus against further herniation, also acts partially in opposition to the sphincter. Thus, varying degrees and types of incompetence, exclusive of primary intrinsic sphincteric muscle failure, may be encountered. Perhaps this explains the paradox of patients with sliding esophageal hiatal hernia, some of whom have reflux whereas others have none. Although gradations in the strength of the sphincter may account for some of these differences, mechanical factors alone may be equally culpable.

It is apparent why hernia repair with reduction into the abdominal

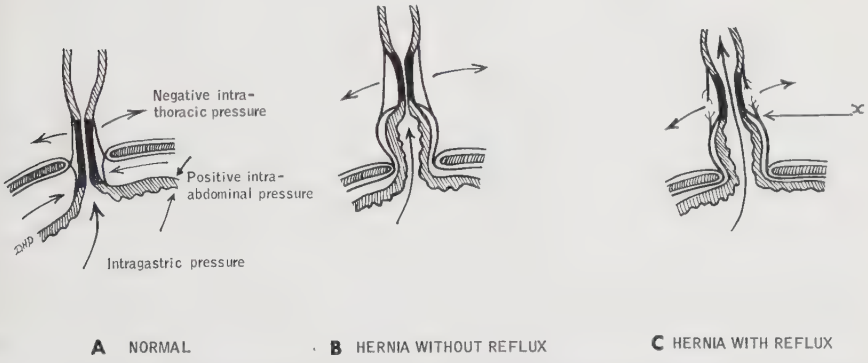

Figure 5. Mechanical and hydrodynamic factors affecting function of the intrinsic sphincter are depicted. The "sphincteric area" is shown in solid black. In *A*, the normal, the sphincter combats only that intragastric pressure which is greater than intra-abdominal pressure since the intra-abdominal pressure imparted to the lumen of the stomach and lower esophagus is cancelled out by an equal pressure against the outside of the abdominal esophagus. In *B*, sliding hiatal hernia is shown. The sphincter has lost the benefit of reinforcement of intra-abdominal pressure, but there is no interference from the phrenoesophageal membrane. In *C*, stretching of the membrane has resulted in an abnormal pull which opposes the action of the intrinsic sphincter and results in reflux.

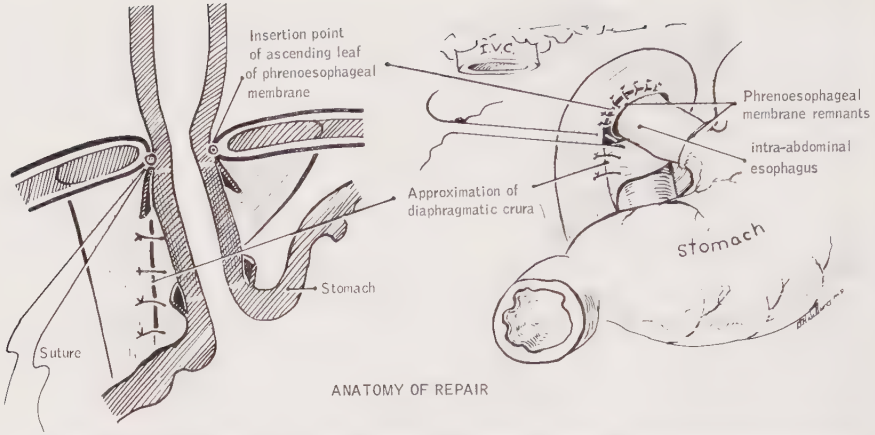

Figure 6. Anatomy of hernial repair. The area of the intrinsic sphincter is shown in cross-hatch shading. The remnants of the ascending and descending leaf of the phrenoesophageal membrane are also shown. The normal intrinsic sphincter functions most effectively in an intra-abdominal position unimpeded by the distracting influence of any structure pulling against it such as might occur if an abnormally inserting phrenoesophageal membrane were utilized in the repair of sliding hiatal hernia; e.g., sutures placed through the phrenoesophageal membrane at point X (Fig. 5, C) to anchor the esophagus to the diaphragm would not deliver the intrinsic sphincter into the abdomen. In addition, the pull of the membrane in holding the lower esophagus down would also oppose contraction of the intrinsic sphincter. The repair shown here stresses reattachment of the hiatus to the esophagus at a level high enough to avoid impingement on the sphincter and ensure delivery of the sphincter into the abdominal compartment.

compartment of the lower esophagus and its contained sphincter will result in control of reflux. By similar analysis, it is also apparent why such a repair in some instances may fail since it is difficult at the time of surgery, especially by the transabdominal route, to determine where the sphincteric area, the squamocolumnar mucosal junction and the original true point of insertion of the ascending leaf of the phrenoesophageal membrane reside. During transabdominal hiatal hernia repair, examination of the lower esophagus internally by means of an operating sigmoidoscope via a gastrotomy has shown that the squamocolumnar, mucosal junction may reside at a higher level than one would have determined this to be by external examination. In this instance, it is possible to narrow the hiatus and resuture the phrenoesophageal membrane remnant to the undersurface of the diaphragm in a manner which does not actually reduce the hernia; i.e., in the sense of having restored the intrinsic sphincter to an intra-abdominal position. Paulson et al., repairing esophageal hiatal hernia by the transthoracic route, have reported excellent results. The outstanding feature of the technique, which was modified after Hayward's description, is utilization of sutures which are placed at the point of insertion of the ascending leaf of the phrenoesophageal membrane as determined from external examination from above. One might easily miss this true point of insertion when working from below the diaphragm. In the absence of an intrinsic sphincter, any

surgical attempt to correct reflux short of jejunal interposition or a valvuloplasty may fail. When the intrinsic sphincter is intact, however, the foregoing mechanical factors are of paramount importance when the surgical treatment of peptic esophagitis is considered.

SUMMARY

The lower esophageal intrinsic sphincter is the primary barrier against gastroesophageal reflux. This sphincter is normally situated within the compartment created by the ascending and descending leaves of the phrenoesophageal membrane and occupies a predominantly intra-abdominal position. In some situations, particularly sliding esophageal hiatal hernia, the effective points of insertion of the phrenoesophageal membrane may be shifted so that interference with intrinsic sphincteric function is produced. This possibility must be recognized and appropriate measures taken to correct it if a satisfactory control of reflux is to be accomplished in the repair of hiatal hernia.

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Details of Interest and Controversy in the Anatomy of the Esophageal Hiatus and Hiatal Hernia

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The treatment of hiatal hernia is still a subject of debate among surgeons, not only because of a difference of personal preference, but also because there is considerable difference of opinion about the physiology of gastroesophageal reflux. The sphincter mechanism is a complex one and as yet there is no widely accepted concept of its mechanism. Recent contributions by Mann and Pisko-Dubienski bring some clarity but not complete agreement.[15, 16] Not only is there difference of opinion about the complicated physiology of this area but there is controversy about the normal anatomy and the anatomy in hiatal hernia.

MUSCULAR ANATOMY OF THE ESOPHAGEAL HIATUS

It is common to read in articles describing methods of repair of hiatal hernia that the author closes the limbs of the right crus in such and such a manner. In actual fact, however, the esophageal hiatus is formed by muscles arising from both the right and left diaphragmatic crura half of the time. In the majority of cases the right crus contributes the principal muscle mass on both margins of the hiatus; but in half of the cases the left crus makes a contribution to either one or both margins of the hiatus and rarely it may provide all the muscle mass about the hiatus. Joining the crura near their origins is the median arcuate ligament which passes anterior to the aorta just above the coeliac axis. This ligament also serves as a point of origin for muscle contributing to the muscular hiatus, either right or left margins or both. The median arcuate ligament is not a constant anatomic structure and in many cases it is very fine or absent. Figure 1 illustrates some of the variations which occur in the muscular relations

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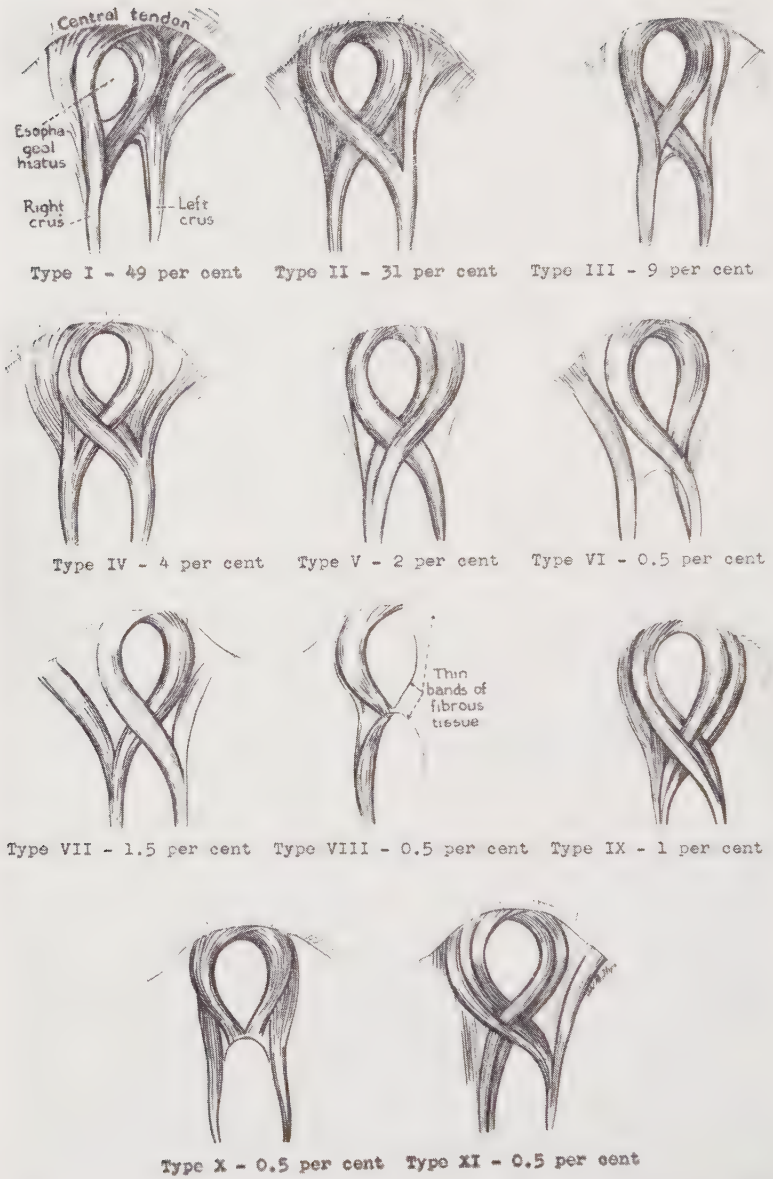
Central tendon
Esophageal hiatus
Right crus
Left crus
Type I - 49 per cent
Type II - 31 per cent
Type III - 9 per cent
Type IV - 4 per cent
Type V - 2 per cent
Type VI - 0.5 per cent
Thin bands of fibrous tissue
Type VII - 1.5 per cent
Type VIII - 0.5 per cent
Type IX - 1 per cent
Type X - 0.5 per cent
Type XI - 0.5 per cent

Figure 1. Variations of the muscular anatomy of the esophageal hiatus. (By permission of J. B. Lippincott Co.[11] Arch. Surg.[12] and West J. Surg. Obst. & Gynec.[13])

about the hiatus. For a detailed description of the variations, the reader is referred elsewhere.[7, 8, 11, 12, 13] Collis et al. have shown that the muscles about the hiatus are enervated by branches of the ipsilateral phrenic nerve even though the muscle origin may be from the contralateral side.[9]

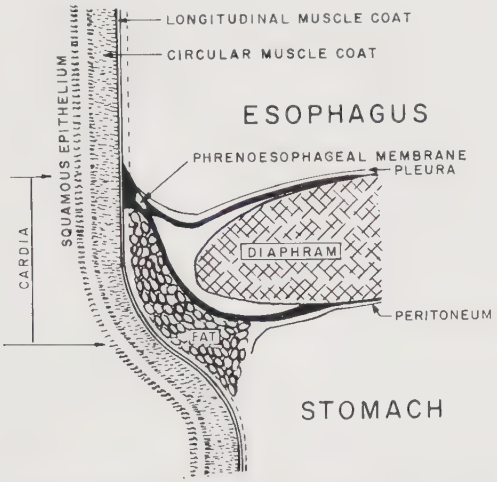

Figure 2. Diagrammatic representation of the origin of the phrenoesophageal membrane. (Modified from Hayward.[10])

THE PHRENOESOPHAGEAL MEMBRANE

The phrenoesophageal membrane is also a structure about which there is considerable debate both as to its anatomy and as to its importance in hiatal repair. The common concept is that it originates from the inferior phrenic endoabdominal fascia. Pisko-Dubienski has recently shown that it is formed by fascial tissue arising from the superior aspect of the diaphragm as well as the inferior aspect.[16] These fascial layers fuse together shortly before inserting into the fascia propria of the esophagus just above the squamocolumnar junction. A few recent dissections of my own confirm this finding. Figure 2, which is a modification of Hayward's illustration, demonstrates this.[9, 10] In young muscular individuals the phrenoesophageal membranes can be a very strong and impressive structure; but in the aged and in hiatal hernia they may be extremely attenuated.

ANATOMY OF HIATAL HERNIA

Sliding Hiatal Hernia

Sliding hiatal hernia is comparable in anatomic principle to the sliding hernias which occur in the inguinal areas in that the posterior aspect of the hernial sac is fixed to a hollow viscus; it differs in that in the groin hernias the hollow viscus remains inside the sac while, at the hiatus, the viscus remains outside.

This hernia is characterized chiefly by the transposition of the esophagogastric junction from a site below the diaphragm to one above. As the junction ascends there is an elongation and often attenuation of the fascial

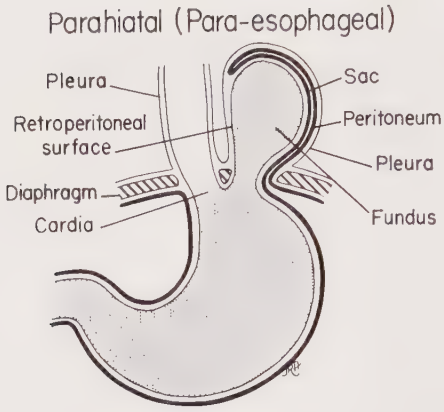

Figure 3. Controversial representation of parahiatal hernia (see text). (By permission of R. H. Sweet and W. B. Saunders Co.[17])

and peritoneal attachments to the hiatus. The size of the muscular hiatus enlarges proportionately to the degree of slide with comparable elongation of both margins of the hiatus. The amount of hiatal enlargement is generally much less than that seen in paraesophageal hernia. A peritoneal sac is formed in the anterior hiatus and separates the central tendon from the stomach. The size of the sac is usually small in comparison to the amount of herniated viscus. As the stomach ascends there is some reflection of peritoneum off the cardia. While the ascending branch of the left gastric artery and its esophageal branches must accompany the cardia in its cephalad migration, its role as a deterrent must be questioned. With this ascent Allison has emphasized that the angle between the esophagus and the stomach is converted from an acute to an obtuse one. The functional significance of this fact is in question but the majority of current methods of repair attempt to restore this acute angle.

Paraesophageal Hernia

Pure paraesophageal hernia without some sliding of the cardia is uncommon. However, the clinical problem of the combined hernia is that of a paraesophageal hernia, so they are considered together.

The principal feature of this hernia is that of a peritoneal sac extending through the hiatus into the mediastinum, usually to the left but occasionally to the right.[6] It is comparable to the indirect inguinal hernial sac into which a hollow viscus may herniate.

There is a difference of opinion as to the hiatal relations in this hernia. Sweet and Berman et al. believe this hernia to be extrahiatal[4, 5, 17] (see Fig. 3). Note the small crural muscle depicted as lying between the esophagus and the herniated fundus. In my experience these hernias have always been intrahiatal and no muscle has been found between the esophagus and fundus. Allison and Barrett support this point of view.[1, 2] Careful attention in the future to this detail both at the operating table and in the morgue will settle this controversy.

The hiatal margins in this hernia may be extremely elongated and attenuated usually involving both margins of the hiatus equally, but occasionally mainly the left margin. Whereas the fascial attachments from the posterolateral hiatus to the cardia remain relatively short, on the anterolateral aspects of the sac they may be stretched beyond recognition.

The sac of this hernia always contains stomach. As the fundus ascends the corpus follows, bringing the attached greater omentum and colon. With further ascent the fundus usually rotates to the right and the greater curvature rolls superiorly into the sac. Less commonly the fundus rotates to the left. The degree of herniation seems to depend on the fixation of the greater curvature to the spleen and the splenic flexure of the colon on the left, and the fixation of the duodenum and hepatic flexure of the colon on the right. In advanced cases almost the entire stomach and much of the transverse colon may be found in the sac.

Rare Types of Hiatal Hernia

Benedini et al. have described a *combined hernia* in which the fundus of the stomach and the peritoneal sac lay posterior to the esophagus[3] (see Fig. 4). This hernia was intrahiatal.

Humphreys et al. reported two operative cases he called *esophago-aortal* hernia in which no crural fibers were found to separate the esophageal orifice from the aorta.[11] One might speculate on the possibility that these hernias might originate from a Type X hiatus (Fig. 1) in which the arcuate ligament may have been weak and separated or may have been absent.

Allison mentions a rare hernia in the hiatal area brought to his attention by Mr. James Davidson in which the defect was extrahiatal.[1] A precise description of this hernia is not available at this writing.

Many of those cases called *congenital short esophagus* are sliding hiatal hernias in which the cephalad migration of the cardia is advanced. This

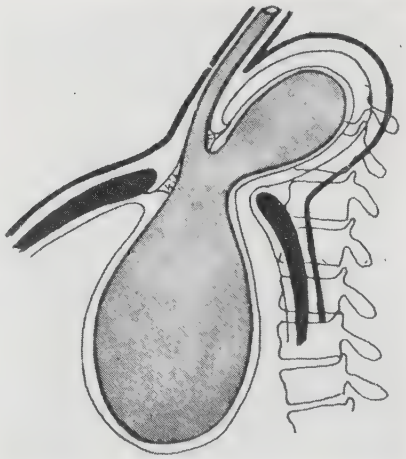

Figure 4. Illustration of rare posterior combined hiatal hernia reported by Benedini et al.[3]

may occur early in life. Humphreys et al. describe a rare case in which the thoracic stomach was supplied by segmental arteries from the aorta.[11] This distinction should be the sine qua non for the definition of congenital short esophagus.

SUMMARY

The important anatomic landmarks of the esophageal hiatus and hiatal hernia have been reviewed. Controversial points have been pointed out. A few rare types of hernias occurring in the hiatal region have been described.

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The Intradiaphragmatic Distribution of the Phrenic Nerve

Surgical Significance

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Considerable interest in the upper portions of the phrenic nerve are indicated by the number of articles in the literature concerning the derivation, anatomy and course of the phrenic nerve within the neck. Until recently,[5] however, the terminal phrenic, as it enters the diaphragm, and its intradiaphragmatic distribution have remained vague. Interest in the proximal phrenic nerve was generated by the utilization of phrenic crush and severance of this nerve in the treatment of tuberculosis. Today, the surgeon's interest in the distal phrenic nerve and its intradiaphragmatic distribution is a result of the development of thoracic surgery, and the importance of combined abdominothoracic incisions which require diaphragmatic incision as well. Obviously, then, it becomes a necessity to determine anatomically and physiologically the pattern of intradiaphragmatic distribution of the phrenic nerve with attention to avoiding unnecessary severance and inactivation of the diaphragm, on the one hand, and the possibility of utilizing controlled segmental paralysis, on the other.

My former associate, Dr. R. X. Maguire, undertook experiments in the laboratory, utilizing the dog as the subject, in the following experimental plan.

EXPERIMENTAL DATA IN THE DOG

Methods of Study

A total of 80 hemidiaphragms (40 right; 40 left) have been studied. All were subjected to the stimulation of the various branches of the phrenic nerve as it terminates in the diaphragm. In addition, following stimulation,

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in 20 isolated hemidiaphragms (10 right; 10 left) the anatomic distribution of the terminal branches of the phrenic nerve was determined by gross dissection in the fresh state.

In the stimulation experiments essentially two methods were utilized:

Isolated Specimens. Ten dogs were sacrificed and the entire diaphragm was removed immediately and completely by severance of its peripheral attachments. Each diaphragm then was pinned about its margin to a large pan of melted paraffin. Thus distortion was avoided. Also it enabled one to observe better the gross contractions following stimulation. The main branches of the phrenic nerve then were dissected immediately and electrically stimulated. Satisfactory responses were obtainable usually for periods of 20 minutes.

In-situ Specimens. An additional 30 dogs were anesthetized with intravenous Nembutal, intubated with an intratracheal tube, and placed on a positive pressure respirator. The thoraces were entered via a transsternal incision in the sixth intercostal space bilaterally. The phrenic nerves were freed of the pericardial sac and the latter's diaphragmatic attachments were severed. The primary branches of the phrenic nerve were isolated and divided. The distal ends were electrically stimulated.

In both types of experiments the branches were stimulated by a two per second stimulus of 1 volt intensity. The anatomic distribution of the nerve and the area of response were recorded on mimeographed charts of the diaphragm, made for this purpose.

Results

Anatomic Findings (Fig. 1). The terminology used for the dog is patterned after that suggested by my former colleague, Dr. R. J. Johnson,[3] from his studies of the human diaphragm.

The phrenic nerve divides into at least two component parts within 1 cm. of the superior border of the diaphragm: one trunk anterior which divides almost immediately into two parts, a sternal (anterior) and an anterolateral branch; and one trunk posterior, which generally turns and runs immediately beneath the parietal pleura toward the posterolateral portion of the diaphragm, usually dividing just before reaching the muscle substance into two branches; a crural (posterior) and a posterolateral. The crural branch on the left is not always constant, as it often appears as a small filament from the main posterolateral branch. Within the muscle substance of the diaphragm the main nerve trunks lie much closer to the central, tendinous portion than to the periphery and the filaments passing to the various muscle groups or bundles *all* radiate costally, none extending toward the central tendon. The main nerve trunks do not appear to lie closer to the peritoneal than the pleural surface. Rather, they lie within a "stratum" approximately midway between the two surfaces. While frequently leaving at right angles to the main nerve trunks, the peripheral filaments usually are given off in such fashion so as to angulate laterad

across several muscle fasciculi. The main trunks could not be traced satisfactorily to the extreme lateral margins of the diaphragm. Furthermore, no anastomotic connections of the antero- and posterolateral branches could be demonstrated.

The right phrenic nerve divides in much the same fashion as the left into two component parts, usually within 1 cm. of the superior border of the diaphragm. The anterior branch divides into sternal and anterolateral components as on the left. The posterior branch runs beneath the pleura as on the left, but generally separates quickly into definite crural and posterolateral components, with greater constancy than on the left. The crural branch is almost always present as a separate and definitive nerve trunk. The distribution of the nerve trunks relative to the muscular portion of the diaphragm is the same on the right as on the left.

Conduction Findings (Fig. 2). The stimulation of either main phrenic nerve trunk caused gross contraction of the ipsilateral hemidiaphragm with relatively little apparent overlap onto the contralateral side. What overlap there was appeared to be limited to the sternal (anterior) segments where some contraction of muscle fasciculi 1 to 2 cm. past the midline could be detected. There seemed to be almost no overlap of the crural (posterior) segments.

STERNAL AND ANTEROLATERAL BRANCHES. Stimulated as one, before or proximal to the point of division into the two components, they exhibited the same degree of anterior midline overlap noted above as well as overlapping laterally into the posterolateral segment for a distance of about 2 cm. The sternal branch has the same overlap across the midline anteriorly, with about 1 cm. overlap into the anterolateral portion. The antero-

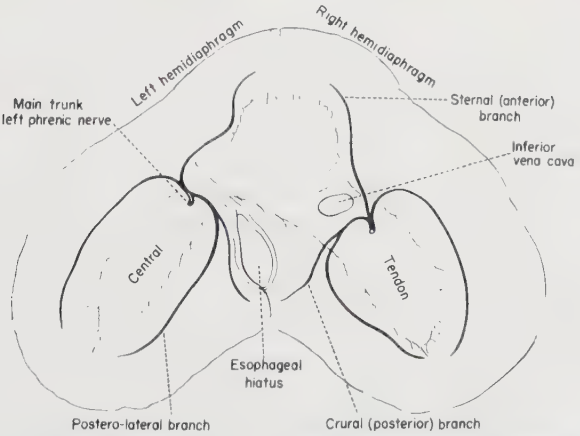

Figure 1. Dog. Viewed from above. The division of the phrenic nerves at the level of the diaphragm and the pattern of distribution of the major branches. This drawing emphasizes the double "handcuff" arrangement of the major branches. The anterolateral and posteromedial branches skirt the central tendon and encircle the dome. (Merendino et al. in Surgery *39:* 189–198, Jan. 1956.)

Figure 2. Dog. Viewed from above. Sphere of influence of each major branch upon electrical stimulation. The stippled areas indicate the segments of the diaphragm which contract upon stimulation of *A*, sternal (anterior) branch, *B*, anteromedial, *C*, posteromedial, *D*, crural (posterior). It will be noted that there is a small overlap in contraction with each branch. Anteriorly this extends into the opposite hemidiaphragm supplied by the right sternal branch. Apparently, there is very little overlap posteriorly in the crural branches. (Merendino et al. in Surgery *39:* 189–198, Jan. 1956.)

lateral branch has close to 2 cm. overlap into the anterior portion and about 2 cm. overlap laterally into the posterolateral portion. Technical difficulties in stimulating the anterior and anterolateral branches separately were considerable as there appeared to be a sort of "scatter" effect. This may have been due to the difficulty of achieving complete electrical isolation of the nerve filaments at the time of stimulation or, perhaps, to the use of too strong an electrical current.

CRURAL AND POSTEROLATERAL BRANCHES. Stimulated as one, proximal to the point of division into the two components, a 1 to 2 cm. overlap into the anterolateral portion was seen, with no appreciable overlap into the opposite crural portion. Due to the difficulty of obtaining a separate crural branch on the left, results were not too satisfactory. On the right, there was less than 1 cm. overlap into the posterolateral portion. On each side there was practically no appreciable overlap across the crus posteriorly.

Therefore, it is to be noted in the dog that the partition of the phrenic nerve as it enters the diaphragm is rather constant. The phrenic nerve characteristically divides into four major branches: sternal, anterolateral, crural and posterolateral. The anterolateral and posterolateral branches skirt the central tendon and tend to approximate each other only at the lateral (midaxillary) diaphragm. This arrangement has been likened to a handcuff. As the course of both right and left nerves is the same, the distribution to the entire diaphragm may be considered to be a double handcuff pattern. While variations do occur, this is a satisfactory schematic concept.

Also, the anterolateral and posterolateral branches are the largest ones,

pursue the longest course, and innervate most of the diaphragm. However, as one might suspect from the definite arrangement of branches, each of the four individual branches of both hemidiaphragms innervates a specific area which contracts when the branch is electrically stimulated. Consequently, the physiologic influence of each branch is segmental in nature with apparently only a minor overlap into adjacent portions of the diaphragm.

DATA ON MAN

The data obtained by the gross dissection of human specimens was carried out by Dr. R. J. Johnson, and the correlations made in the operating room were made by the author (K.A.M.). These two studies complemented each other well, indicating a reasonable correlation with the information obtained in the experimental laboratory with the dog.

Gross Anatomy

The right phrenic nerve reaches the diaphragm just lateral to the inferior vena caval aperture and at times may use that aperture as a route through the diaphragm. The left phrenic nerve reaches the diaphragm just lateral to the left border of the heart and in a somewhat more anterior coronal plane than is the case for the right nerve. Instead of piercing the central tendon as does the right, however, the left nerve passes through the muscular portion of the diaphragm anterior to the central tendon.

Typically, the phrenic nerve (either the right or the left) divides at the level of the diaphragm, or a centimeter or two above it, into several terminal branches. Of these branches two or three are commonly very fine and are distributed to the serosal surfaces of the diaphragm, the pleura above, and the peritoneum below. These pleural and peritoneal twigs, when present as discrete branches, supply only the central serosal areas.

The greater mass of the dividing phrenic nerve continues entirely, or part of the way, through the diaphragm for its subsequent distribution as muscular rami. A few scattered filaments are given off from these muscular rami to the subjacent peritoneum. The muscular rami, here in the area where they pierce the diaphragm, vary from two to four in number. Fundamentally, there are four main rami and four major areas of distribution. A reduction in number is brought about by two branches sharing a common trunk for a short distance.

In the most common pattern three muscular branches arise directly from the phrenic nerve. One is directed anteromedially toward the sternum. Another is directed laterally anterior to the lateral leaf of the central tendon. The third branch is directed posteriorly and shortly divides into a branch which runs laterally posterior to the lateral leaf of the central tendon and into a branch which runs posteriorly and somewhat medially

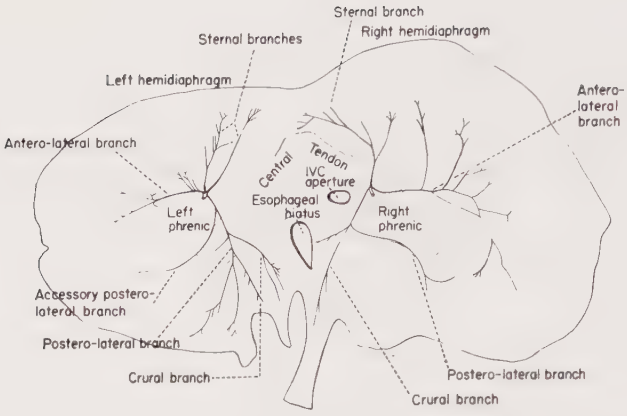

Figure 3. Human being. Actual findings in a gross dissection of a human diaphragm. This drawing is according to scale and viewed from above. (Merendino et al. in Surgery *39:* 189–198, Jan. 1956.)

to gain the region of the crus. Thus a pattern of four major branches is seen with the two more posterior branches often having a short common trunk. These four branches may be named, respectively, the sternal or anterior branch, the anterolateral branch, the posterolateral branch, and the crural or posterior branch.

Figure 3 portrays a specimen which fairly closely follows this typical or "standard" plan. In the preparation of this figure the strongly curved surface of the diaphragm was projected onto a plane surface by extending the muscular portion out radially from the central tendon. The entire diaphragm was drawn life size. Conversion of the curved diaphragm into a plane figure inevitably results in some magnification of the peripheral portion insofar as circumferential measurements are concerned. It will be noted, from both Figure 3 of an actual specimen and from Figure 4, a schematization of the pattern in the human diaphragm, that the anterolateral and posterolateral branches of both the right and the left phrenic nerves form curves embracing the domes of their respective hemidiaphragms like the arms of a pair of manacles.

These muscular rami were often found to be submerged in the muscle substance rather than lying exposed on the inferior surface as commonly stated in textbooks. After the peritoneum and endoabdominal fascia were removed, many specimens revealed only segments of these muscular branches as the rami wended their way through the muscular substance, here and there appearing briefly at the surface or being seen only as terminal twigs supplying the peritoneum. As a crude estimate it might be said that 50 per cent of the course of the muscular rami is a submerged one requiring the division of muscle fasciculi to expose the nerve.

Operating Room

In patients subjected to a thoracic incision the terminal portion of

the phrenic nerve has been isolated. By the nature of most clinical conditions, a left thoracotomy incision is more frequently used than a right. Consequently, most information gained from the operating room has been of the left phrenic nerve. However, there is no indication from the previous work presented that such information is not equally valid for the right phrenic nerve.

Anterior and posterior divisions of the phrenic nerve are readily demonstrable at the diaphragmatic level. Usually, three or four branches are present. By their size and anterior or posterior position, these are identifiable as sternal, anterolateral, posterolateral and crural. The anterolateral and posterolateral divisions are always the larger trunks. If there is a single large branch anteriorly, by dissecting it a few centimeters the sternal branch can be identified as it courses toward the sternum. Similarly, if the crural branch is not immediately apparent, by following this larger posterolateral branch it will be observed to leave this main trunk and be directed toward the esophageal crus. On occasion, the crural branch was not identifiable. Undoubtedly, it takes off the posterolateral branch more distally than the dissection was carried out.

In patients subjected to pneumonectomy, these branches have been isolated and divided. Each has been repeatedly crushed in order to determine the area of contraction. In general, the area of contraction follows the identical pattern observed experimentally in dogs.

Practical Considerations

Because of the rapid diminution in size of the branches of the phrenic nerve after they enter the diaphragm, it would be impractical to attempt

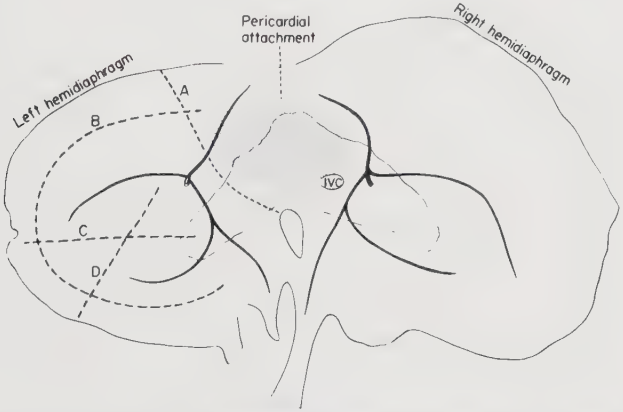

Figure 4. Human being. Schematized drawing indicating the "safe" areas in the diaphragm where incisions may be made without any significant effect on diaphragmatic function. *A*, The diaphragmatic component of a combined abdominothoracic incision extending down into the esophageal hiatus. *B*, Circumferential incision. *C*, *D*, Incisions extending from lateral (midaxillary) and posterior costal areas into the central tendon (from above). See text. (Merendino et al. in Surgery *39:* 189–198, Jan. 1956.)

to delineate the peripheral distribution of each individual nerve. Furthermore, as implied earlier, this is unnecessary. It is sufficient to know the four branches and the "handcuff" pattern created by the anterolateral and posteromedial divisions.

The Placement of Diaphragmatic Incisions. This information indicates that there are "safe" areas in the diaphragm where incisions may be made with relative impunity and assurance that the branches of the phrenic nerve in the diaphragm will be preserved. If severance of a branch appears to be necessary, the denervation knowingly may be restricted to a small segment of the diaphragm.

Circumferential incisions anywhere in the periphery of the diaphragm result in little if any dysfunction of the diaphragm. Incisions through the central tendon, almost as far medially as the entrance of the phrenic nerve, can be made without the interruption of any major branch. Lateral or transverse incisions from the midaxillary line medially likewise result in almost no paralysis. This incision is of use in transthoracic splenectomy.

Combined abdominothoracic incisions, if properly placed, would not appear to significantly affect diaphragmatic function. When the abdominal and thoracic walls have been incised with severance of the costal cartilage, the diaphragmatic incision must extend medially and downward between the entrance of the phrenic nerve into the diaphragm and the pericardial attachment to the diaphragm. The diaphragm then can be opened down to and including the apex of the esophageal hiatus. In this fashion only the relatively unimportant sternal division will be severed. All other nerve components remain intact. When one begins to separate the ribs by forceful retraction, the attachment of the phrenic nerve and the pericardiacophrenic vessels with their fatty components must be separated from the pericardium for 14 to 15 cm. proximally. Otherwise, the nerve filaments within the nerve sheath may be ruptured. When this precaution is observed, full exposure can be obtained with the phrenic nerve attached to the diaphragm which retains normal action. There is only the mild annoyance of working about the phrenic nerve, which is a small price to pay for its preservation (Fig. 4). In combined incisions for splenorenal or portacaval anastomoses, full exposure can be obtained by incising the diaphragm circumferentially in its peripheral aspects, thus avoiding phrenic nerve injury.

DISCUSSION

The importance of normal diaphragmatic action is appreciated by the fact that, during quiet breathing, the diaphragm accounts for 60 per cent of the total air ventilated.[4] By measurement at death, unilateral diaphragmatic paralysis decreases the volume of the hemithorax up to 33 per cent.[1] An average decrease of 33 per cent in vital capacity in humans has been observed following phrenic exeresis.[7] Pulmonary function studies indicate

that a phrenicopraxia (14 days postoperative) was observed to produce a 21 per cent loss of vital capacity and maximum breathing capacity, and an 18 per cent loss of oxygen uptake on the crushed side.[2] Extrapulmonic effects subsequent to phrenic interruption include the slowing of the venous return and gastrointestinal disturbances. The author has observed one case of severe esophagitis on a reflux basis following left pneumonectomy which was accompanied by a phrenic crush (left), which resulted in an adverse arrangement of the stomach with the esophagogastric junction apparently producing incompetence of the sphincteric mechanism.

Despite the fact that there is considerable optimism regarding the complete restoration of normal diaphragmatic function subsequent to phrenic crush, in one study of 157 cases some degree of permanent paralysis existed in approximately one-half and a total loss of diaphragmatic function in one-fifth of the cases,[6] thus highlighting the importance of avoiding needless sacrifice or damage of the phrenic nerve at any level.

Today, constant stress is placed upon lowering the morbidity and mortality of surgical procedures. Considerable attention is paid to preventing the preventable death. The improvements in ancillary supporting fields tend to encourage boldness on the part of the surgeon. Extensive major surgical procedures are offered to a more aged patient population than heretofore. In contrast to the surgery of some years back, death of the postsurgical patient is less frequently the result of a single gross error. It is an impression that more frequently today the patient dies not of a single complication, but often of the effects of multiple small complications which individually would not be lethal but when compounded result in a fatality. This presentation is an attempt to isolate one of these factors. There can be little doubt that the immediate loss of pulmonary function with its adverse effect on venous return and the gastrointestinal tract may be poorly tolerated by the borderline-risk patient. Few would deny the importance postoperatively of a good effective cough and the increased incidence of pulmonary atelectasis and bronchopneumonia secondary to an elevated atonic diaphragm. In the aged these factors may represent the difference between life and death.

It has been shown that phrenic interruption with diaphragmatic paralysis is a preventable complication. Furthermore, the concept of controlled segmental paralysis is a usable maneuver in the surgeon's hands.

SUMMARY

The muscular fibers of the phrenic nerve, in both dog and man, are arranged in a fundamental pattern of four major rami: a sternal (anterior), an anterolateral, a posterolateral, and a crural (posterior). By coalescence of the proximal portions of these major branches variations occur in the number of these branches at the diaphragmatic level.

The schematic pattern is best described as a double "handcuff" arrangement with the anterolateral and posterolateral branches being the main components skirting circumferentially and lateral to the dome. By electrical stimulation the diaphragmatic contraction is noted to be segmental. The segment affected is dependent upon the anatomic area supplied by the stimulated branch. There appears to be a small overlap with each branch and anteriorly across to the opposite diaphragm.

By the proper placement of diaphragmatic incisions, the phrenic branches can be avoided and function of the diaphragm preserved. This should obviate certain postoperative complications attributable to phrenic paralysis. Furthermore, if segmental paralysis of the diaphragm seems indicated, this can be accomplished by crushing individual branches rather than the entire phrenic nerve.

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The Embryologic Approach to the Surgical Management of Carcinoma of the Upper Stomach

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Techniques for the removal of malignancies of the gastrointestinal tract are designed to resect the pathways of lymphatic drainage in continuity with the primary neoplasm wherever this is anatomically possible. For example, in removing carcinomas of the large intestine, it is mandatory to redevelop a large area of the mesentery and include with it the lymph nodes and blood vessels to their origins from the superior or inferior mesenteric arteries as an en bloc excision.

Malignant lesions of the stomach, however, are usually excised by a subtotal gastric resection that differs slightly if at all from the gastrectomy commonly performed for benign peptic ulcer. The greater and lesser omenta, all accessible lymph nodes along the curvatures, and frequently the spleen are included in the resection. However, most of the areas of lymphatic drainage from the stomach are beyond the limits of these procedures. These lymphatic pathways are largely retroperitoneal, and the lymph nodes are inextricably bound to the pancreas and to the origins of the celiac and superior mesenteric vessels deep to the usual plane of dissection of the gastrectomy commonly performed for cancer (Fig. 1).

The proximal portion of the stomach drains primarily into the left gastric and splenic nodes and thence into the left, middle and right suprapancreatic groups of nodes. The flow from the antral and pyloric portions of the stomach is primarily into the left gastric, the right gastric (suprapyloric), the right gastroepiploic, the subpyloric, and the main superior mesenteric nodes. Both the antral and the fundic portions of the stomach, then, drain into the left gastric nodes. Unfortunately, the left gastric and

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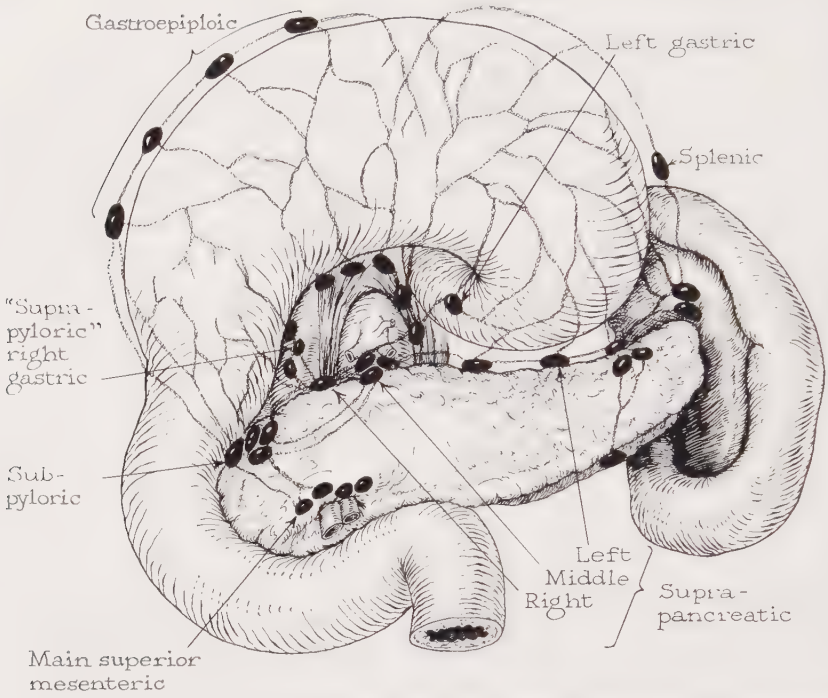

Figure 1. The principal lymphatic vessels and nodes of the stomach. The stomach has been rotated superiorly.

suprapancreatic nodes can be excised only by the inclusion of the tail of the body of the pancreas in continuity with the gastric lesion, since the pancreas is actually contained within the mesentery of the stomach.

It is significant that excision of certain groups of lymph nodes about the stomach has been emphasized as being of importance. The greater omentum, for example, is removed to effect the complete excision of the gastroepiploic nodes. Similarly, the importance of splenectomy to excise the splenic nodes in the gastrosplenic ligament has been emphasized. However, these nodal areas are actually peripheral to the main lymphatic pathways and are of secondary importance since their efferent vessels drain ultimately into the celiac group of nodes, the vortex of the metastatic whorl. From the standpoint of lymphatic dissemination of malignancy, the nodes at the base of the celiac axis are at the neck of the funnel for lymphatic flow from the proximal stomach. The flow, therefore, must be pinched off at this point because beyond it the spread is to the periaortic lymph nodes which lie beyond the scope of curative resection. Because the gastric lymphatic vessels enmesh the body of the pancreas, it is reasonable that the body of the pancreas must be removed if the "wide removal of lymphatic-bearing tissues" is to be accomplished. Metastatic deposits of tumor cells in these nodes have been confirmed by pathological study of resected specimens which have included the body of the pancreas.[1, 3, 5]

In planning an operation designed to remove these lymphatic pathways, it is important to note those regions to which no drainage is directed. In vivo studies with injections of dye have demonstrated that the lymphatic vessels of the stomach do not primarily traverse the head of the pancreas nor do they primarily reach the retroperitoneal tissues around the vena cava.[6] These findings correspond to those of Berry and Rottschaffer,[2] who found no positive lymph nodes in those areas during Whipple operations performed as a clinical experiment in a small series of patients with advanced carcinoma of the stomach. The transection of the pancreas at the junction of its neck and body should, therefore, be sufficient for removal of the major lymphatic pathways of the stomach.

The anatomical basis for such a resection rests entirely on concepts acquired from a consideration of the embryology of the foregut. In the early embryonic development, the stomach is part of a straight tube suspended parallel to the aorta by the dorsal mesogastrium. Within the dorsal mesogastrium, the spleen and body of the pancreas are developing (Fig. 2). As the liver enlarges, the stomach and its mesogastrium are displaced to the left side of the abdomen. The left lateral leaf of the mesogastrium becomes applied to the posterior abdominal wall and the opposed

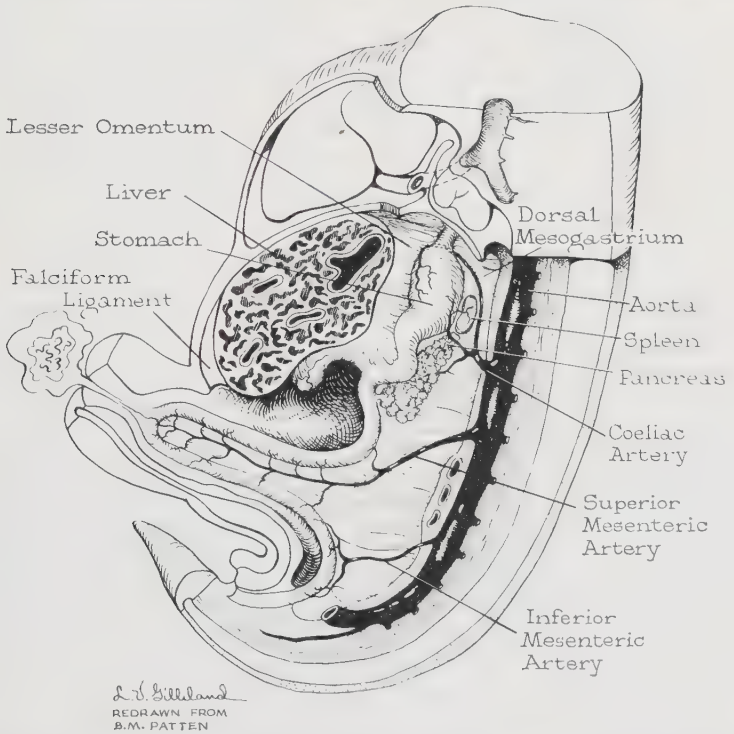

Figure 2. Sagittal section of the human embryo prior to intestinal rotation illustrating the relation of the pancreas and spleen to the stomach and the primitive mesogastrium.

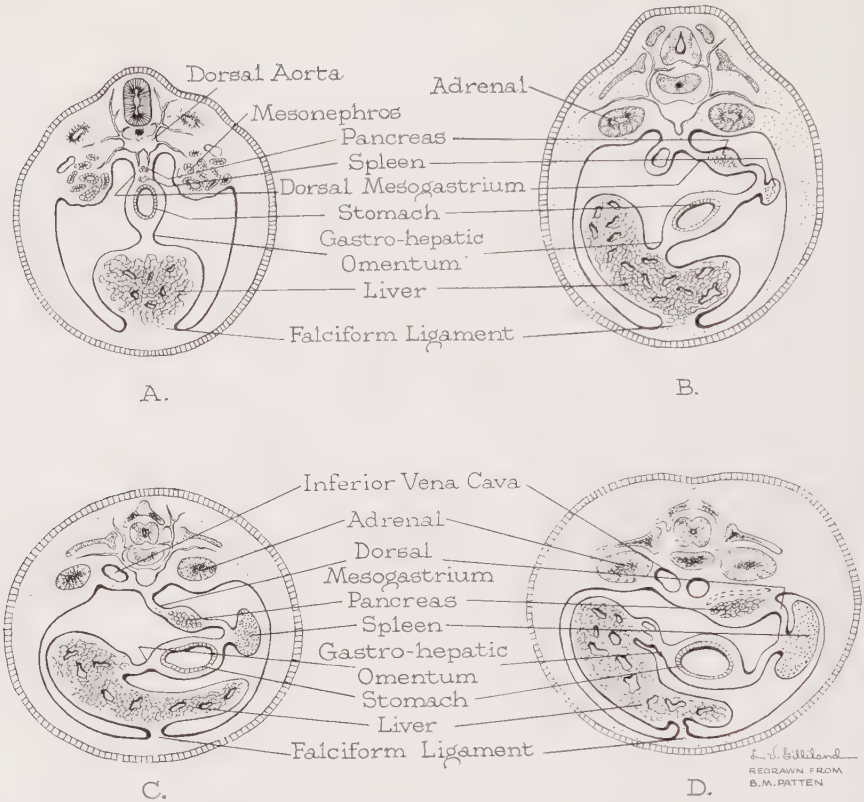

Figure 3. Cross sections through the foregut of the human embryo to illustrate the progressive stages (*A*-*D*) of development of the spleen and body of the pancreas in the primitive mesogastrium and the ultimate fixation of the pancreas to the posterior abdominal wall. The plane of fusion is the dotted area behind the pancreas (*D*).

mesothelial surfaces are resorbed, leaving a potential areolar plane of cleavage. The body and tail of the pancreas thus become fixed in a retroperitoneal position. Anterior to the pancreas, the pleated surfaces of the mesentery, instead of fusing, become the walls of a space, the omental bursa. This space communicates with the general peritoneal cavity through an opening at its dextral extremity, the foramen of Winslow. Four layers of embryonic tissue result at the apex of the pleated formation at the hilus of the spleen. Of these, two form the gastrosplenic ligament and two the splenorenal ligament of the adult anatomy. The potential space posterior to the pancreas represents an embryologic plane of fusion across which there is no structural continuity (Fig. 3). This plane may be reopened at operation without injury to any organ or major blood vessel.

Knowledge of this embryonic development has practical application in operative procedures. When removing the upper stomach and its lymphatic pathways, this primitive plane is available for reopening without transection of the lymphatic pathways within the mesogastrium. If the

continuity of that mesentery is violated, lymph nodes into which the stomach drains will remain outside the scope of the operative procedure (Fig. 4).

In the distal foregut, the anlage of the entire biliary system and pancreas arise as buds from the entoderm of the second portion of the duodenum (Fig. 5). The body of the pancreas appears as an outpouching from the duodenum and its growth continues within the leaves of the mesogastrium. The head of the pancreas first appears as a separate evagination of the duodenum as a portion of the entodermal complex that is to differentiate into the liver and the biliary system. Throughout its further development, the pancreatic head maintains this intimate relationship to the common bile duct within the mesoduodenum.

The two embryologically distinct rudiments of the pancreas persist up to the stage of intestinal rotation. During this later event, the midgut passes counterclockwise posterior to the superior mesenteric axis and the head of the pancreas comes to lie behind the coronal plane of the portal vein while the body of the pancreas remains anterior to that plane. Grant[4] has graphically described this movement of the two pancreatic rudiments as closing on the portal vein "like a book on a book-mark." A communication is established between the two ducts of the pancreas and thereafter the lower duct (duct of Wirsung) conducts the main volume of pancreatic juice into the duodenum. However, the upper duct (duct of Santorini) usually persists and drains into the duodenum through an ostium cephalad to that of the major pancreatic duct.

Because of its embryonic development, the pancreas may be regarded

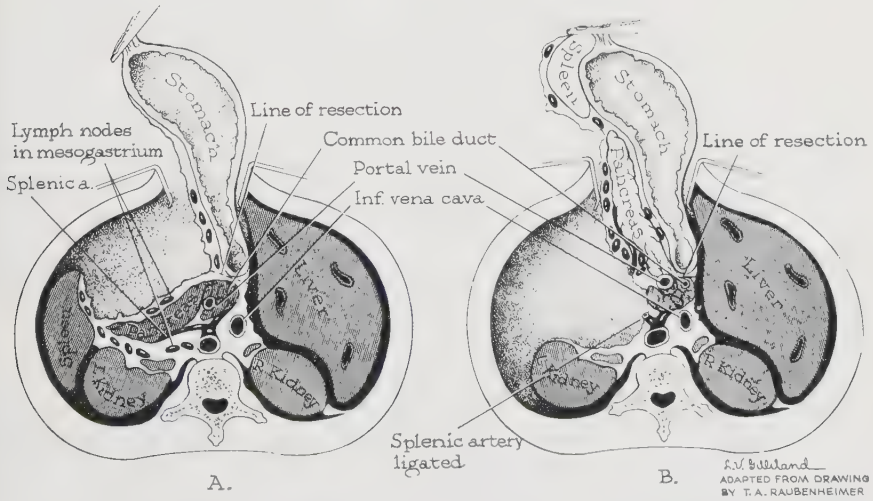

Figure 4. Cross sections illustrating a conventional gastrectomy (*A*) which interrupts the primary lymphatic pathways of the stomach and leaves behind many of the principal lymphatic depots that would be removed by an "extended gastrectomy" (*B*).

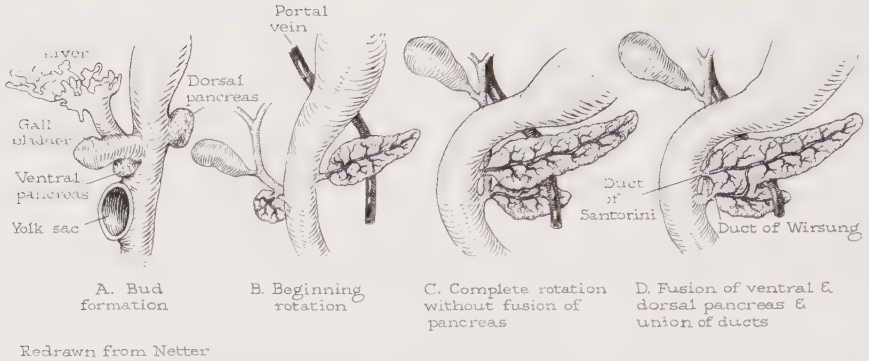

Figure 5. The head and body of the pancreas arise as separate buds from the foregut. See text.

surgically as a single organ composed of two separate units. Each unit, for example, has an individual blood supply. The head of the pancreas is supplied through the arterial arcade developed between the superior and inferior pancreaticoduodenal arteries; the body of the pancreas receives its blood from the splenic artery. In most cases, the main pancreatic duct courses through the head of the pancreas so that the egress of pancreatic secretion from this portion of the gland cannot be interrupted by removal of the body of the pancreas.

Of prime importance, from the standpoint of lymphatic flow from the stomach, is the fact that the body of the pancreas lies within the *mesogastrium*, whereas the head of the pancreas is in the *mesoduodenum*. Thus, the two parts of the gland are in different coronal planes of the body. The head of the pancreas is in a plane posterior to the portal vein; the body of the pancreas is anterior to the portal vein, and the two are bridged through the neck of the pancreas. The latter may be considered a hinge connecting the two parts of the pancreas. If, at operation, mobilization of the proximal portion of the stomach is conducted through the plane of embryonic fusion and if the stomach is detached at its upper end and the left gastric and splenic blood vessels are ligated at their origins, the entire stomach with the intact mesogastrium may be rotated out of its bed on this hinge. If the hinge is then disconnected by division across the neck of the pancreas, complete excision of the mesogastrium and, thereby, the primary regional lymphatic pathways of the stomach is effected.

Carcinomas of the proximal portion of the stomach are often of considerable size and extent since they easily escape detection in their early stages. Many of them have, therefore, reached the stage where fixation to surrounding structures has occurred. Erroneously, they may be considered nonresectable because of a severe degree of fixation posteriorly. In reality, the body of the pancreas intervenes between the stomach and the area posterior to it and may actually prevent direct extension of the primary gastric malignancy into the retroperitoneal tissues. This is an extremely

useful surgical concept. If the embryologic plane of tissue fusion behind the pancreas and spleen is redeveloped, the removal of large adherent primary gastric lesions cannot only be accomplished with ease but en bloc excision of the entire lymph-bearing area contained within the mesogastrium can be carried out as well. By the adaption of these anatomical and embryological concepts to the management of patients with carcinoma of the proximal stomach, many lesions that would have been considered inoperable only a few years earlier can now be removed.

These concepts are particularly applicable to malignant lesions of the proximal stomach for two reasons. One is the ability to accomplish an en bloc resection of the primary lesion and the lymphatic drainage areas just discussed. Secondly, a portion of the distal stomach can be preserved for the re-establishment of gastrointestinal continuity by means of an esophagogastrostomy. Even with preservation of the distal stomach, groups of lymph nodes in the suprapyloric and infrapyloric regions can be excised although not en bloc.

TECHNIQUE

The operative procedure for malignancies of the proximal stomach, applying these embryological and anatomical principles, consists of the en bloc excision of the distal esophagus, the proximal two-thirds of the stomach, the spleen, the tail and body of the pancreas, and the entire greater omentum together with the regional lymphatic vessels and nodes.

The abdominal cavity is explored initially to determine operability of the gastric lesion. If resection is possible, the abdominal incision is extended into the left thorax through the seventh intercostal space and the diaphragm is divided through the esophageal hiatus.

Mobilization of the lesion is begun by dividing the splenorenal ligament, a move that allows the spleen to be rotated medially. The embryologic plane of fusion posterior to the pancreas is thereby entered and carried medially along the retroperitoneal area by gentle finger or other blunt dissection. The development of this plane is carried to the point where the inferior mesenteric vein crosses behind the pancreas to join the splenic and superior mesenteric veins to form the portal vein. The entire greater omentum is excised (Fig. 6).

The peritoneum overlying the cardioesophageal junction is incised and the vagus nerves are divided. This maneuver allows approximately 2 inches of the esophagus to be drawn down into the abdomen. Since the supradiaphragmatic lymphatic areas may be involved in the presence of a malignant lesion of the proximal stomach, dissection of the lymphatic-bearing areas is extended to the level of the inferior pulmonary vein. The lymphatic and areolar tissues overlying the diaphragmatic crura are removed and the lesser omentum is divided flush with the inferior surface

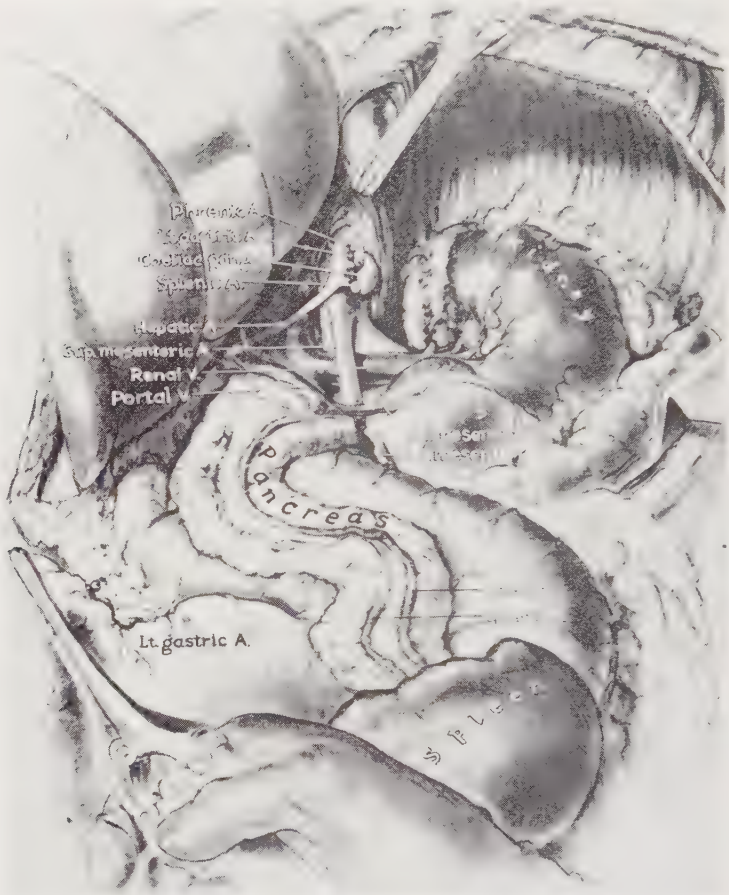

Figure 6. The esophagus has been divided and the dissection has been carried to the base of the celiac axis. The vessels are divided at their origins. The stomach, spleen and pancreas are mobilized as a unit by blunt dissection along the retroperitoneal embryologic plane of fusion.

of the liver. The left gastric artery is ligated at its origin from the celiac axis. The splenic artery is then easily identified and similarly ligated at its origin. The splenic vein is ligated just proximal to its confluence with the portal vein. The pancreas is divided at the junction of its body and head and sutured with interrupted catgut sutures.

The stomach is divided at the junction of the body and antrum, preserving the right gastroepiploic and right gastric vessels. The subpyloric, suprapyloric and right suprapancreatic nodes overlying the gastroduodenal artery and the head of the pancreas as well as the main superior mesenteric nodes may be readily removed (Fig. 7).

Gastrointestinal continuity is established by mobilization of the distal stomach and proximal duodenum. A two-layer anastomosis is performed between the esophagus and the anterior wall of the gastric remnant.

Interrupted cotton sutures are used throughout for the anastomosis. A Heineke-Mikulicz pyloroplasty is established (Fig. 8).

DISCUSSION

Experience with 36 patients undergoing this procedure since 1956 has indicated that it has some merit in the management of the patient with malignant lesions of the proximal stomach. There have been no direct operative deaths and only three patients have succumbed from complications. One of these expired from the effects of acute empyema from a breakdown at the esophagogastric anastomosis; one had unexplained severe

Figure 7. Mobilization has been completed and the specimen "hinges" on the neck of the pancreas. The stomach is transected in the antral area. The entire mesogastrium containing all the principal nodal depots and the intervening lymphatic vessels has been excised en bloc. Removal of the pyloric nodes can be accomplished without injury to the vascular structures.

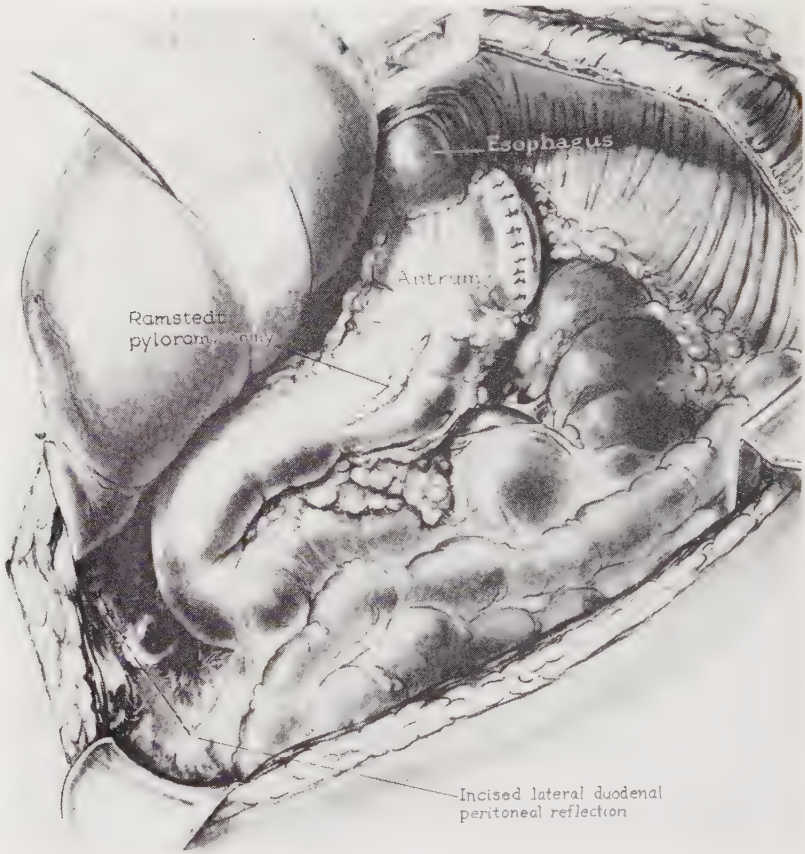

Figure 8. Reconstruction by esophagogastrostomy. A pyloroplasty of the Heineke-Mikulicz type is usually performed rather than the Ramstedt pyloromyotomy illustrated here.

dysphagia and died from inanition. The third patient expired from a sudden pulmonary embolus one month following discharge after a smooth postoperative course.

It is impressive to note the ease with which an en bloc dissection can be accomplished through embryologic tissue planes despite the presence of extremely large posteriorly invasive tumors. In some patients, direct invasion of the pancreas through the posterior wall of the stomach was present. More often than not, however, the pancreas acted as a barrier to further fixation and invasion and the retropancreatic embryologic plane of fusion remained open and easily dissectable. The utilization of this plane for dissection allows the resection of many lesions heretofore considered inoperable. The operative procedure is based on sound anatomical, physiological and embryological principles, all of which are in keeping with current concepts of cancer surgery.

SUMMARY

The surgically important features of the anatomy and embryology of the stomach and pancreas have been correlated in the design of an operative procedure for the treatment of cancer of the upper stomach. The basic aim of this technique is the complete removal of the gastric mesentery containing the primary pathways of lymphatic drainage from the stomach. The accomplishment of this objective requires subtotal removal of the stomach, the spleen, the body of the pancreas, and the greater and lesser omenta. By focusing on the removal of the gastric mesentery as a primary objective, the block of tissue to be resected is sharply defined.

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A New Anatomic Approach to the Problem of Incomplete Vagotomy

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INTRODUCTION

As a general rule, surgeons think in terms of regional anatomy rather than systemic anatomy. Regional or surgical anatomy works better when applied to operations. In the case of vagotomy however, regional anatomy has proved not to work. Incomplete vagotomy occurs because we have been trapped by the fallacy inherent in emphasizing regional anatomy at the expense of systemic anatomy.

The trap is the diaphragm, a structure that has nothing whatsoever to do with the embryology or anatomy of the vagus nerves. At the esophageal hiatus the vagal system may occur in the form of any one of three components—the esophageal plexus or the trunks or the truncal divisions. Therefore, when we use the diaphragm as the landmark for regional anatomic study of the vagal system, and fail to recognize the vagal system as either the esophageal plexus or the trunks or the truncal divisions, our anatomic concept can be nothing more than one of unorganized variations.

From this concept stems the problem of incomplete vagotomy. So many alleged variations of the vagal system at the hiatus have been described that they are generally viewed as unpredictable and inevitable pitfalls. Vagotomy is accordingly looked upon as a hit-or-miss procedure bound to result in a certain rate of failure. Thus incomplete vagotomy is not only a problem from the standpoint that it occurs, but also from the standpoint that its occurrence is generally considered an unavoidable misfortune.

The purpose of this paper is to challenge the problem of incomplete vagotomy with a new anatomic concept—new not in the sense that something has just been discovered, but new in the sense that it departs from the conventional regional anatomic concept. If we remove the diaphragm, we remove the trap and with it the futility of trying to conceive of the

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vagal system as it exists only at the hiatus. We now have an unobstructed view that permits us to study systemic anatomy from thorax to abdomen. The esophageal plexus, the vagal trunks, and the truncal divisions to the abdominal viscera soon fall into a rather uniform system that explains and organizes all the variations into one basic anatomic pattern. But of even more importance, we see that certain pathways are, in certain respects, constant. And when we replace the diaphragm, the basic pattern and the constants remain. It is this systemic anatomic concept of constants, rather than the regional anatomic concept of variations, that provides the basis for achieving complete vagotomy with anatomic accuracy and consistency.

THE BASIC PATTERN

Below each pulmonary hilum, the left and right vagus nerves branch and communicate with each other to form the *esophageal plexus.* Depending upon the number of branches and communications, this plexus varies from a few large fibers in its simplest form to many small fibers in its most complex form. Contrary to rather popular opinion, the variable complexity of the esophageal plexus precludes any guarantee that complete vagotomy can *always* be accomplished by the transthoracic technique (particularly when the plexus exists as a rete).

The fibers of the esophageal plexus unite to form *two and only two vagal trunks,* one *anterior* and the other *posterior* to the esophagus. As a result of communications in the esophageal plexus, each trunk contains fibers from both vagus nerves. However, the anterior trunk contains more fibers from the left vagus nerve than from the right, and the posterior trunk contains more fibers from the right vagus nerve than from the left. Thus the two vagal trunks emerging from the esophageal plexus are by no means identical to the two vagus nerves entering the plexus.

Each of the two vagal trunks gives rise to two primary divisions. The anterior trunk divides into *hepatic* and *anterior gastric* divisions, and the posterior trunk divides into *celiac* and *posterior gastric* divisions. These truncal divisions represent the primary pathways for the distribution of vagal innervation to the abdominal viscera (Fig. 1).

THE VARIATIONS

By superimposing the diaphragm on the basic pattern, we introduce variations. However, keeping the basic pattern in mind, we see that the variations are for the most part only pseudo-variations, which may be classified into two types of variable positions and two types of variable numbers (the so-called "multiple vagus nerves").

Variable Position Type I—Due to Embryologic Rotation of the Stomach

Prior to embryologic rotation of the stomach, the lesser curve faces ventrally. One vagal trunk lies on the left of the esophagus, and the other vagal trunk lies on the right. After rotation, the lesser curve faces the right, and the left and right trunks assume anterior and posterior positions respectively (Fig. 2). The mnemonic is LARP ("left anterior" and "right posterior").

But LARP is confusing. At the level of the distal esophagus and cardia, the vagal trunks rotate the full 90 degrees with the stomach. Therefore, at this distal level, the right posterior trunk always assumes a right posterior position, but the *left* anterior trunk always assumes a *right* anterior position. Furthermore, up at the level of the hiatus, LARP is

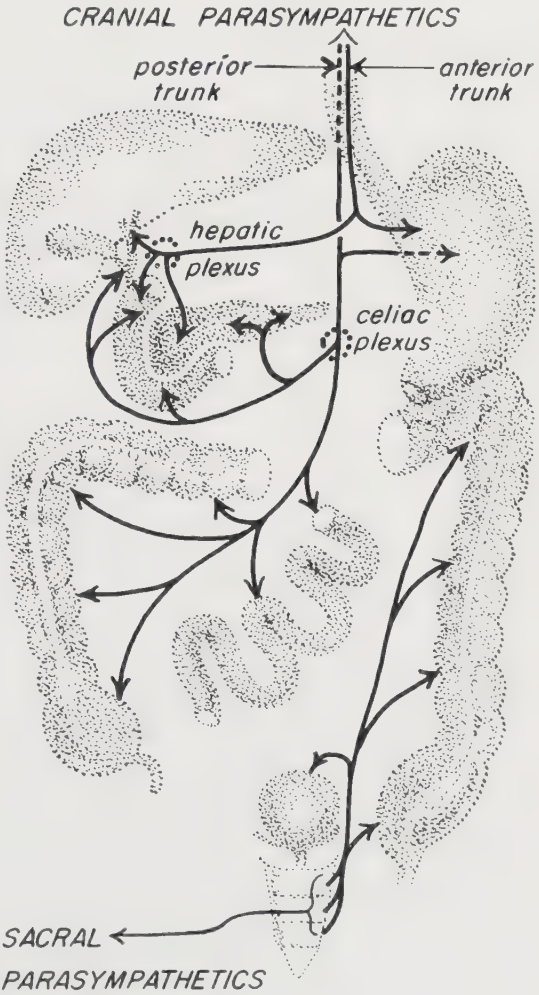

Figure 1. Peripheral vagal distribution. The hepatic division from the anterior vagal trunk supplies the parasympathetic components of the autonomic hepatic plexus (dotted circle). From the hepatic plexus, hepatic vagal fibers are distributed to the gallbladder, biliary tract, head of pancreas and proximal duodenum.

The celiac division from the posterior vagal trunk goes to the celiac plexus, from which its fibers accompany the superior mesenteric artery to the pancreas and entire midgut. The hind gut is innervated by the sacral parasympathetics.

The anterior and posterior gastric divisions do not traverse any plexus. They travel directly and alone to the stomach.

(From Griffith: West. J. Surg. Obst. & Gynec., 1962.)

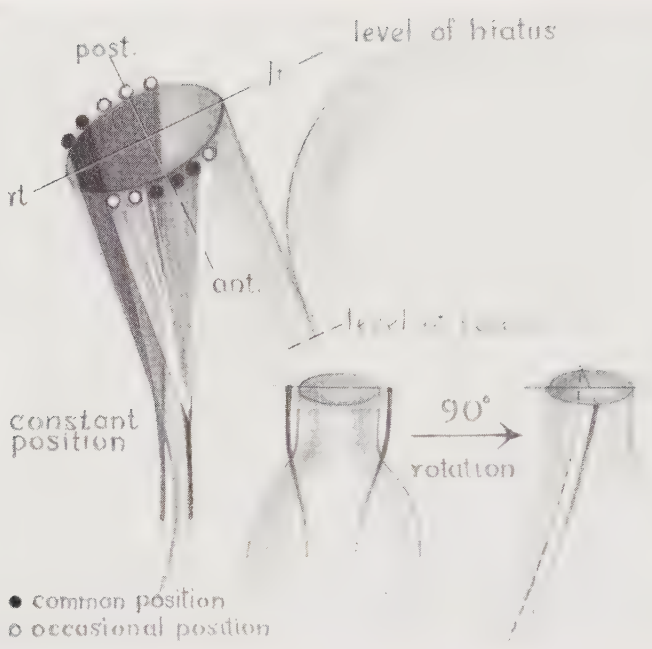

Figure 2. Embryologic rotation of stomach and vagal trunks.

even more confusing. At this higher level the vagal trunks are quite removed from the effects of rotation, and therefore usually rotate through a variety of degrees to assume a variety of positions. At the hiatus the left anterior trunk may lie on the right, and the right posterior trunk may lie on the left (Fig. 2).

The moral of the story is this. LARP applies only to embryology. If applied to surgical anatomy, LARP becomes meaningless and even misleading. The positions of the vagal trunks at the hiatus are so variable that we cannot describe them in any terms or mnemonics applicable to all positions. Furthermore, we do not use the terms "right anterior" and "right posterior" to describe the constant positions of the trunks along the distal esophagus. But in regard to vagotomy, we might do better if we did. Therefore, so long as a mnemonic seems to be in order, I would suggest RARPADE ("right anterior-right posterior-at distal esophagus"). At least RARPADE emphasizes the constant positions of the vagal trunks in the only place they are constant.

Variable Position Type II—Extreme Dorsal Position of Posterior Trunk

In addition to its variable left and right positions at the hiatus, the posterior vagal trunk lies in variable ventral and dorsal positions between the esophagus and aorta. When the posterior trunk lies close to the esophagus, as it usually does, we can feel the trunk quite readily with the finger

encircling the esophagus. But when the trunk lies well away from the esophagus, in an extremely dorsal position adjacent the right diaphragmatic crus and aorta, the encircling finger may miss the trunk completely. This technical error is probably the most frequent cause of incomplete vagotomy (Fig. 3).

"Multiple Vagus Nerves" Type I—The Low Lying Esophageal Plexus

The fibers of the esophageal plexus unite to form the two vagal trunks at variable levels. Usually the trunks are formed at variable distances

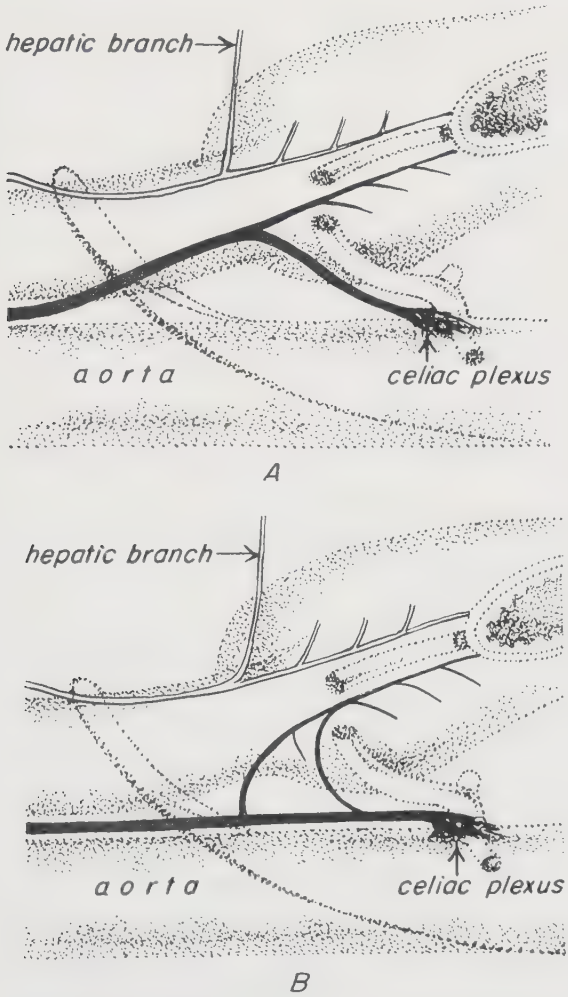

Figure 3. Variable dorsal positions of posterior trunk and celiac division.

A, The posterior trunk is adjacent to the esophagus. The celiac division runs close to the left gastric artery on its way to the celiac plexus.

B. The posterior trunk and celiac division lie in an extreme dorsal position closely applied to the diaphragmatic crus. (From Griffith: West. J. Surg. Obst. & Gynec., 1962.)

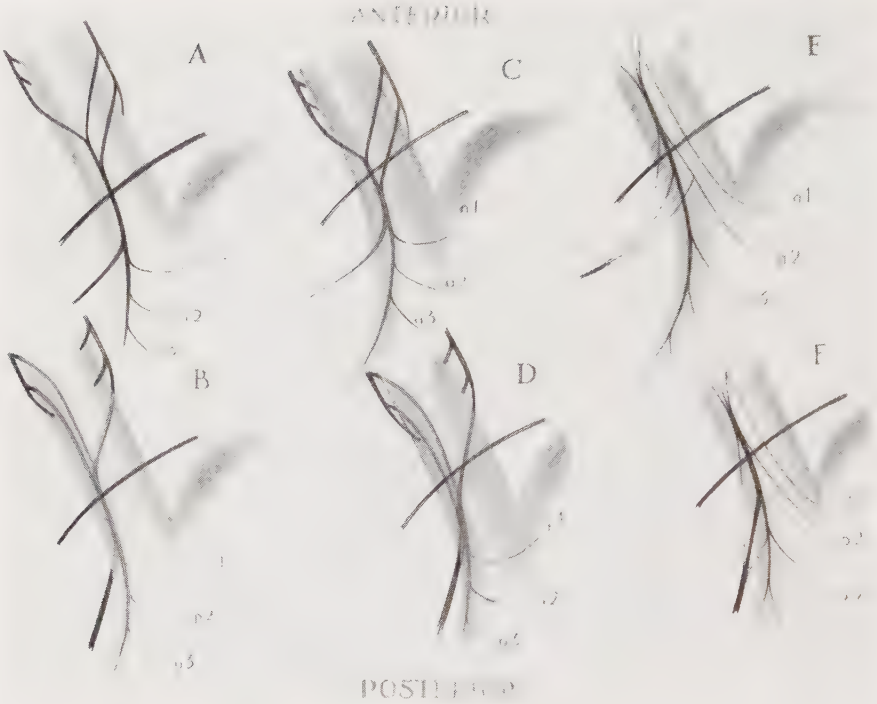

Figure 4. "Multiple vagus nerves."

A, B, The vagal system at the hiatus consists of the anterior (*A*) and posterior (*B*) trunks.

C, D, The vagal system at the hiatus is the esophageal plexus.

E, F, The vagal system at the hiatus consists of the truncal divisions. Note the suprahiatal origin of gastric vagal fibers *a1* and *a2* to the anterior fundus, and *p1* and *p2* to the posterior fundus.

above the hiatus, and pass through the hiatus as two single trunks. Occasionally, however, the esophageal plexus extends below the hiatus, in which case the "multiple vagus nerves" at the hiatus are in reality the esophageal plexus. The two trunks are formed below the hiatus (Fig. 4).

"Multiple Vagus Nerves" Type II—The Suprahiatal Origin of Primary Truncal Divisions and Terminal Branches

Most commonly the vagal trunks give rise to their primary divisions well below the hiatus. But occasionally the trunks divide above the hiatus. The resulting "multiple vagus nerves" at the hiatus are separate hepatic, celiac and gastric divisions. This sets the stage for either or both selective hepatic and selective celiac vagotomy if the gastric divisions are missed.

More commonly however, "multiple vagus nerves" occur when the most proximal of the terminal gastric branches to the fundus arise not from the primary gastric division, but separately from a trunk or even the esophageal plexus above the hiatus. The same holds true for a small

hepatic or celiac fiber (Fig. 4). In this pattern the two main trunks are always present. The terminal branch to the gastric fundus always lies to the left of the trunks, and the hepatic and celiac twigs always lie to the right of the trunks.

THE CONSTANTS

The Hepatic Division

Throughout its course from the anterior vagal trunk to the porta hepatis (Fig. 1), the hepatic division lies directly beneath the anterior peritoneal layer of the lesser omentum. Although it may exist as one large branch or several small closely approximated fibers, the hepatic division usually consists of three to four strands that at first casual glance resemble fibrous peritoneal thickenings. The hepatic fibers run parallel to, and within two or three fingerbreadths of, the attachment of the lesser omentum to the liver. This portion of lesser omentum adjacent the liver is free of fat in even the most obese patients, and therefore the hepatic division is never obscured by fat. It can always be seen when the lesser omentum is exposed and stretched out by upward retraction of the left lobe of the liver and downward retraction of the stomach. The clearest view of the hepatic division is obtained by visualizing the caudate lobe of the liver through the stretched out, fat-free transparent lesser omentum, and seeing the gray opaque hepatic fibers within the lesser omentum contrasted against the dark background of the caudate lobe.

Identification of the hepatic division goes hand in hand with ascertaining the dangerous presence of an aberrant left hepatic artery from the left gastric artery. This aberrant vessel occurs in about 10 to 15 per cent of the population. When present it runs just below the hepatic vagal division in the lesser omentum to the porta hepatis. Because an aberrant left hepatic artery may carry all of the arterial blood to the left lobe of the liver, inadvertent injury of this artery during gastric resection has been reported to result in fatal necrosis of the left hepatic lobe (Friesen, 1957).

The Celiac Division

Two constant features characterize the celiac division. First, it is the largest of all the primary truncal divisions, and always exists as a large fiber approximating the size of a heavy No. 0 or No. 1 suture material. Second, the celiac division always goes to the celiac plexus. Because of its size and course, the celiac division is perhaps best conceived as the continuation of the posterior vagal trunk.

However, the exact position of the celiac division in the ventral-dorsal plane—i.e., between the esophagus and left gastric artery ventrally, and the right diaphragmatic crus and aorta dorsally—is not constant (Fig. 3). In its usual ventral position, the celiac division hugs the dorsal aspect of

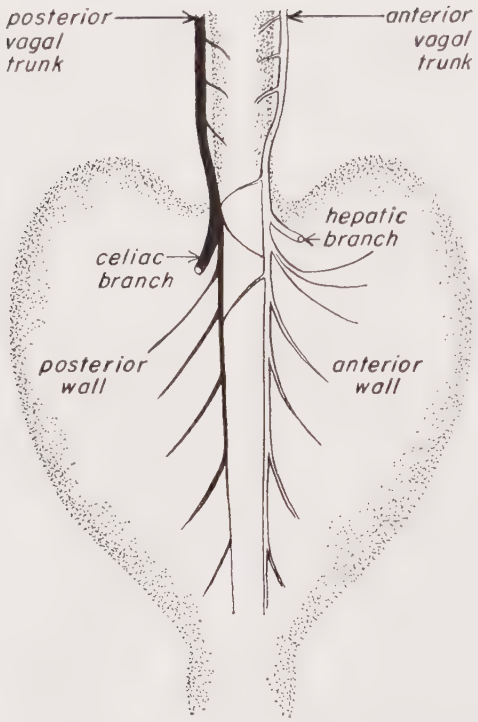

Figure 5: Vagal innervation of the stomach. The stomach has been opened along the greater curve and laid flat (mucosal surface down). Fibers connecting the anterior and posterior gastric divisions along the lesser curve constitute the so-called gastric plexus. Not diagrammed is the occasional twig from the anterior trunk contributing to the celiac division, and a similar twig from the posterior trunk to the hepatic division. All fibers in this diagram can be demonstrated by gross dissection. (Modified from McCrea, 1924.)

the left gastric artery within the pancreatic-gastric peritoneal fold. In this position, inadvertent and unknown selective celiac vagotomy is often the result if the celiac division is ligated and transected with the left gastric artery during gastric resection. In its unusual dorsal position, however, the celiac division lies well removed from the left gastric artery and descends to the celiac plexus closely applied to the right diaphragmatic crus and aorta.

The Gastric Divisions

As described in Figures 3 and 4, the origins of gastric vagal fibers vary. Figure 3,*B* shows the unusual occurrence of posterior gastric fibers arising from the celiac division and reaching the stomach in company with the left gastric artery (Jackson, 1948). Figure 4 shows the occasional occurrence of individual terminal fibers to the proximal stomach originating separately from the trunks or esophageal plexus.

Despite these variations in origin, however, vagal innervation of the stomach may be considered constant in that all gastric fibers go to the right aspect of the cardia and proximal lesser curve (RARPADE). No fibers from either the gastric divisions or the hepatic and celiac divisions go to the *greater* curve by circuitous routes through the hepatic and celiac plexuses.

The anterior gastric division continues downward from the anterior

trunk to assume its constant position alongside the right aspect of distal esophagus and cardia. From here it descends along the anterior aspect of the lesser curve as the principal anterior gastric nerve (of Latarjet). Individual terminal branches from this principal anterior gastric nerve pass to the anterior wall of the stomach and innervate it in a segmental manner from fundus to pylorus. The posterior gastric division from the posterior vagal trunk innervates the posterior wall of the stomach in a similar manner (Fig. 5).

SURGICAL APPLICATION

Essentially three techniques of conventional truncal vagotomy have evolved through Dr. Dragstedt's experience: (1) transthoracic, (2) transabdominal and (3) transabdominal supradiaphragmatic through a counter-incision of the diaphragm (Dragstedt, 1961). In either case, the vagal system is exposed at or just above or below the hiatus.

As a means of accomplishing complete vagotomy, these conventional techniques present two real drawbacks. The first drawback concerns variations. Exposure of the vagal system in the region of the hiatus necessitates a dissection with variable numbers of nerves (Fig. 4) in variable positions (Figs. 2 and 3). These variations predispose to the most common of all technical errors –failure to include all vagal fibers when the esophagus is initially mobilized and encircled. The excluded fibers are not brought into the surgical field, but are displaced by the encircling finger into areolar tissues that later become stained with blood. In this displaced position and obscured from view, the excluded fibers may never be found. Thus is the die for incomplete vagotomy cast at the outset.

This error of excluding vagal fibers cannot be recognized by the custom of submitting segments of vagal fibers as surgical specimens. Even if the pathologist does frozen sections and reports that all of the fibers are indeed nerves, he cannot determine that they represent *all* the nerves to the stomach any more than he can determine by examining a hernia sac that it has been ligated high enough. Nor can this error be recognized by the physiologic test for completeness of vagotomy contributed by Burge and Vane (1958). This test entails manometric measurement of the rise in intragastric pressure when the stomach contracts in response to vagal stimulation by an electrode designed to encircle the esophagus. Vagotomy is deemed complete when intragastric pressure no longer rises in response to vagal stimulation. If the electrode does not include all vagal fibers, however, this test is not valid. (In fairness to this test, however, we shall refer to it later.)

The only means of assuring completeness of vagotomy is providing an adequate field of dissection in which we can recognize (1) the variations and consequent technical errors, and (2) the fibers that go to the stomach.

This brings us to the second drawback of conventional techniques—failure to provide an adequate field of dissection. A dissection confined to or by the hiatus is not only inadequate for differentiation between the esophageal plexus and trunks and truncal divisions, but is inadequate for identification of all gastric vagal fibers as well. Accurate identification of the peripheral destination of any vagal fiber encountered at or just above or below the hiatus is all too often an anatomic impossibility. The only possible way to determine its peripheral destination is to dissect it distally and see where it goes. Therefore, to draw a comparison, just as we expose enough of the biliary tract to gain anatomic proof that the duct we transect is the cystic duct from the gallbladder and not the common duct from the liver, so must we expose enough of the vagal tract to gain anatomic proof that the fibers we transect are the fibers to the stomach and not the fibers to the hepatic and celiac plexuses.

If we apply RARPADE, we avoid the pitfalls in the region of the hiatus. By starting the dissection at the distal esophagus and cardia, we are in an area where the vagal system is much more constant. We can identify the hepatic and celiac divisions with anatomic precision and accuracy, and use them as guides to locate the trunks and gastric divisions in an orderly manner. With this identification of the fibers that go to the stomach, we can perform our vagotomy with anatomic assurance that it is complete.

This dissection with the hepatic and celiac and gastric divisions is also the basis for selective gastric vagotomy. Despite the controversy concerning the physiologic and clinical significance (or insignificance) of preserving vagal innervation to viscera other than the stomach, the chief advantage of the selective technique is the consistently successful result of complete vagotomy of the stomach. Furthermore, this advantage holds true when the selective technique is applied to either selective gastric or conventional total abdominal vagotomy.

TECHNIQUE

Application of constant landmarks at the distal esophagus and cardia guarantees inclusion of all vagal fibers when the esophagus is encircled. The landmark on the right is the hepatic division. By incising the lesser omentum to enter the lesser peritoneal space both above and below the hepatic division, the hepatic division is defined and isolated in order to be certain that we have included all vagal fibers on the right. No fibers go to the stomach to the right of the hepatic division. The landmark on the left is the angle between the gastric fundus and esophagus. By snipping the peritoneum over this angle, and introducing the finger into the crotch between the fundus and esophagus, we can be certain of including all vagal fibers on the left. No gastric fibers run to the fundus to the left of or across

this angle. By working through the lesser omentum below the hepatic division on the right, and through the angle between the fundus and esophagus on the left, we feel for the posterior landmark—the celiac division. Although its position between the left gastric artery and the diaphragmatic crus is variable (Fig. 3), we can always identify the celiac division by palpation because of its size and course to the celiac plexus. By encircling the celiac division we insure inclusion of all gastric fibers posteriorly. A soft catheter or drain replaces the encircling fingers to maintain exposure. Now, certain that we have included all fibers to the stomach, the operation proceeds according to the choice of selective gastric or total abdominal vagotomy. (Because Mr. Burge encircles all gastric vagal fibers in this manner, his physiologic test for completeness of vagotomy is valid in his hands).

Downward traction on the stomach stretches out the gastric vagal fibers to facilitate their identification and dissection. In thin individuals this maneuver brings into view the anterior gastric division just beneath the peritoneum adjacent the distal esophagus and cardia. Fat obscures the anterior gastric division in obese patients, but with traction on the stomach it can always be palpated as a taut string in its constant RARPADE position.

By definition, the anterior vagal trunk is that fiber which bifurcates into the hepatic and anterior gastric divisions. Therefore, we may trace these divisions proximally to the bifurcation, and thus quickly and accurately identify the anterior trunk for total truncal vagotomy.

For anterior selective vagotomy, the incision extends from the opening in the lesser omentum just beneath the hepatic division on the right, to the opening in the peritoneum at the angle between the fundus and esophagus on the left. This results in transection of the anterior gastric division plus any and all terminal fibers to the proximal stomach that may have arisen from the trunk or esophageal plexus above. It is made between clamps because small branches of the ascending esophageal arteries from the left gastric artery are transected as well. The anterior vagal trunk and hepatic division are dissected upward, so that the entire anterior surface of the esophagus is bared from the hiatus to cardia.

In performing anterior selective vagotomy, we must take into consideration the fact that some but not all dissections reveal one or more tiny fibers from the hepatic division or plexus descending alongside the right gastric vessels to the pylorus (McCrea, 1924; Mitchell, 1940, 1959; Franksson, 1948; Jackson, 1948). Their function in man is unknown. Our studies in dogs (Stavney et al., 1963) have failed to elicit any motile response of the pylorus when the hepatic division is electrically stimulated. Therefore, hepatic fibers descending to the pylorus may be afferent. At any rate, Burge (1960) sections the gastrohepatic ligament containing these descending hepatic vagal fibers and the right gastric vessels to supplement anterior selective vagotomy.

Anterior selective vagotomy is technically easy. If only from the standpoint of technique, it is recommended over anterior truncal vagotomy because the resulting exposure after the selective method facilitates posterior vagotomy. In the previous description of how the celiac division is located by palpation when the esophagus is initially encircled, it is readily admitted that this maneuver, like all technical maneuvers, becomes easy only by doing it. Therefore, if the celiac division is not found by this maneuver, it must be found after anterior vagotomy is completed. Location of the celiac division after anterior selective vagotomy in a field that extends from hiatus to cardia is much easier than in the more confined field of dissection that usually follows anterior truncal vagotomy.

The celiac division is the guide to the posterior trunk and the posterior gastric division. It is traced proximally and freed from the entire abdominal esophagus. By this technique, location of a posterior gastric division arising at or just below or above the hiatus is relatively easy. On the other hand, demonstration of a posterior gastric division arising at the lower level of the left gastric artery is sometimes technically difficult—particularly in obese patients. Because this distal origin is common, and because gastric fibers may arise even more distally to reach the lesser curve with the left gastric artery (Fig. 3, *B*), posterior selective vagotomy entails ligation and transection of the left gastric artery adjacent the lesser curve. From this point upward the cardia and esophagus are freed of lesser omentum so that the posterior trunk and celiac division are completely separate from the entire abdominal esophagus and cardia. In thin patients this dissection is relatively easy, but is difficult in the obese.

By this technique, the only gastric fibers that can possibly remain are those of suprahiatal origin (Fig. 4). These fibers lie in the same fascial plane as the trunks, and therefore are not to be found within the esophageal muscle. Twigs from the trunks or esophageal plexus that plunge into the esophageal wall innervate the esophagus, and do not continue downward within the muscle to innervate the stomach. These esophageal fibers can be freed only by avulsing them out of the esophageal muscle, which may be a factor in postoperative dysphagia. Dissection of the esophagus in search of gastric vagal fibers is therefore not done with the theory that the only guarantee of complete gastric vagotomy is transection of the esophagus. Instead, any remaining gastric vagal fibers are found exterior to the esophageal muscle within its fascia propria. They may be lifted off the esophageal wall, traced to the cardia to prove that they are gastric fibers, and excised.

SUMMARY AND CONCLUSIONS

1. The occurrence of incomplete vagotomy is generally considered unavoidable because of innumerable variations of vagal pathways. This

concept of variations stems from regional anatomic studies of the vagi at the hiatus, and is fallacious because no embryologic or anatomic relationship between the vagi and the hiatus exists.

2. If the vagal system is studied as a system without relating it to the diaphragm, the variations may be organized into a rather constant anatomic pattern. This pattern consists of three components: (1) the esophageal plexus, (2) two and always two trunks, and (3) four and always four truncal divisions —the hepatic, the celiac, and the anterior and posterior gastric divisions.

3. The hepatic and celiac divisions are anatomic constants. Although the origins of the gastric divisions vary, the gastric divisions are constant from the standpoint that they always innervate the stomach along the lesser curve.

4. Incomplete vagotomy by conventional techniques of total abdominal (truncal) vagotomy occurs because (1) the vagal system is exposed in the region of the hiatus where it may exist as the esophageal plexus or the trunks or the truncal divisions (the variations), and (2) the field of dissection is too limited to permit accurate identification of either the specific component of the vagal system exposed or the fibers that go to the stomach.

5. Dissection with the constant truncal divisions at the distal esophagus and cardia is the basis for the technical accomplishment of complete vagotomy because the field of dissection is adequate for accurate identification of all gastric fibers. This dissection is also the basis for selective vagotomy of the stomach.

6. Regardless of the controversy concerning the *physiologic* rationale of selective gastric vagotomy, the chief advantage of the selective technique is the *anatomic* assurance that vagotomy of the stomach is complete. The selective technique may be applied to either total abdominal or selective gastric vagotomy for consistently successful results of complete and total vagal denervation of the stomach.

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Surgical Anatomy of the Pancreas

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GROSS ANATOMY

The pancreas extends transversely across the upper abdomen behind the stomach. Its head lies in the concavity of the duodenum and its tail in the region of the hilus of the spleen. The anterior surface is partially hidden from view by the attachment of the transverse colon to the head and by the attachment of the transverse mesocolon to the inferior portion of the body and tail. The pyloric region of the stomach and the first portion of the duodenum overlap the head of the pancreas anteriorly. Posteriorly, the head of the pancreas lies against the inferior vena cava, both renal veins and the right renal artery. The body and tail lie upon the aorta, left adrenal and left kidney.

The junction of the upper left portion of the head of the pancreas with the body is called the neck or waist. This constricted and narrowed area is rarely more than 3 to 4 cm. wide. The superior mesenteric vein and artery lie just behind the neck and are also enclosed behind by a posterior extension of the lower left portion of the head (uncinate process).

The pancreas is a relatively fixed organ. Some degree of superior and inferior movement may occur, however, and pancreatic tumors are occasionally seen to move during respiration. The fixation of the pancreas accounts for its vulnerability to blunt trauma, which may fracture it posteriorly across the rigid vertebral column. The position and fixation of the pancreas also explain the fact that aortic pulsations are directly transmitted to the organ. As a result, pancreatic tumors are sometimes confused with aneurysms of the aorta, especially since pain in the back is common to both conditions. Because of the close relationship of the head of the pancreas with the common bile duct and duodenum, these organs are often obstructed by tumors or pancreatitis. It is indeed surprising that obstruction of the mesenteric vessels, especially the superior mesenteric vein, does not occur more often in association with diseases of the pancreas, since

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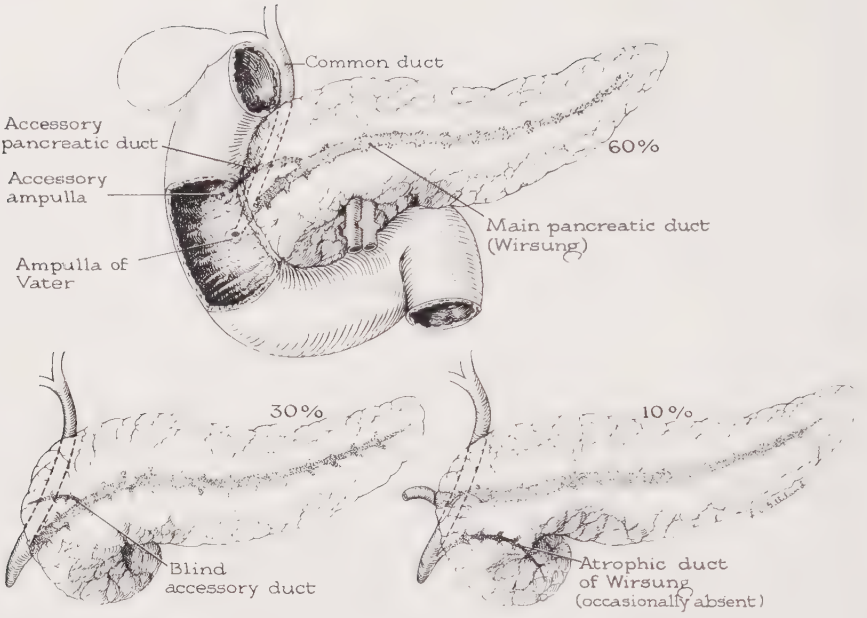

Figure 1. Anatomic configuration of intrapancreatic ductal system. Note lack of communication between two ducts in 10 per cent of cases.

these vessels are almost entirely enclosed by pancreatic tissue (the neck of the pancreas anteriorly and the uncinate process posteriorly). The relatively slow growth of neoplasms, which allows time for the development of collateral venous drainage, and the early death of patients with cancer caused by metastatic spread probably explain this paradox.

Ductal System

The main pancreatic duct (duct of Wirsung) begins at the tail of the pancreas and extends to the right, at or slightly superior to a point halfway between the superior and inferior surfaces of the pancreas. It lies somewhat closer to the posterior than to the anterior surface of the organ. When the duct is extremely dilated, as often occurs in tumors of the head of the pancreas or in chronic pancreatitis, it may be palpable as a wormlike or ballottable structure within the pancreas. Its tributaries enter almost at right angles along its entire course. In the head of the pancreas the major duct turns inferiorly and joins the common bile duct at the major papilla of Vater. The minor duct of Santorini lies in the head of the pancreas in a much more ventral and therefore in a surgically more vulnerable plane than the duct of Wirsung. This duct begins at its junction with the main duct in the neck of the pancreas and terminates in the minor papilla, located about 2 cm. proximal to the major papilla and 7 cm. distal to the pylorus. Tiny accessory ducts emptying directly from the pancreas into the intrapancreatic portion of the common bile duct have been described by Cross.[2]

Many variations in the normal anatomy of the ductal system have been described (Fig. 1). Configuration of the ducts is normal in only 60 to 70 per cent of the population. In about 30 per cent of cases, although a minor duct of Santorini exists it does not communicate with the duodenum. Reich[7] has recently found that this anatomic arrangement is more frequent in patients with duodenal ulcer, and he has suggested that the lack of alkaline pancreatic juice in this region may make the duodenum more susceptible to peptic ulceration. In 5 to 10 per cent of humans, the duct of Santorini is the sole duct draining the pancreas into the duodenum. In such cases injury to the duct of Santorini, especially during gastrectomy, may cause severe hemorrhagic or relapsing pancreatitis. Since the gastroduodenal artery always passes behind the duodenum proximal to the minor papilla, this vascular landmark may be useful as an indication that further distal dissection of the duodenum may be extremely hazardous. Occasionally, a deep penetrating duodenal ulcer erodes into the duct of Santorini, and the surgical management of this complication is dependent upon the knowledge of the ductal anatomy.

In the second decade of life the major pancreatic duct is 3 to 4 mm. in diameter. As aging progresses the ducts become larger, and in persons over the age of 70, ducts 5 to 6 mm. in diameter are commonly encountered in the normal pancreas.

Blood Supply

The arterial supply to the pancreas is remarkably constant (Fig. 2). Anterior and posterior arcades supply the head of the pancreas; each arcade derives a superior component from the gastroduodenal artery and an

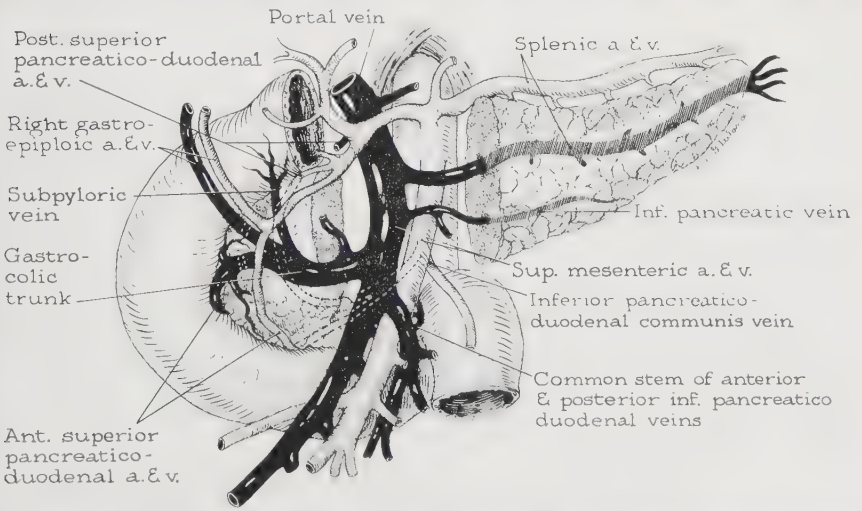
Portal vein
Post. superior pancreatico-duodenal a.& v.
Splenic a.& v.
Right gastro-epiploic a.& v.
Subpyloric vein
Inf. pancreatic vein
Gastro-colic trunk
Sup. mesenteric a.& v.
Inferior pancreatico-duodenal communis vein
Common stem of anterior & posterior inf. pancreatico duodenal veins
Ant. superior pancreatico-duodenal a.& v.

Figure 2. Arterial supply and venous drainage of the pancreas. Note particularly the anatomic importance of the gastrocolic venous trunk in defining the inferior portion of the neck.

inferior component from the superior mesenteric artery. The anterior-superior pancreaticoduodenal and anterior-inferior pancreaticoduodenal arteries join to form the anterior arcade; the posterior-superior pancreaticoduodenal and posterior-inferior pancreaticoduodenal arteries form the posterior arcade. This arrangement occurs in almost 100 per cent of cases. The anterior arcade constitutes virtually the only major group of blood vessels on the anterior surface of the pancreas, an important surgical consideration. The inferior pancreatic artery, which is nearly always constant, runs along the postero-inferior surface of the pancreas in intimate contact with the organ. Its origin is variable; it may start from the superior mesenteric, anterior-superior or inferior pancreaticoduodenal, or the superior pancreatic artery. The superior pancreatic artery is present in from 50 to 90 per cent of cases and arises from the splenic, hepatic, superior mesenteric or celiac arteries. It lies along the posterosuperior portion of the neck and body of the pancreas. The body and tail of the pancreas are also supplied by branches from the splenic and left gastroepiploic arteries.

The venous drainage of the pancreas follows the course of the arteries quite closely, but the veins lie superficial to their arterial counterparts. Anterior and posterior venous arcades are always present around the head of the pancreas. The confluence of the right gastroepiploic, anterior-superior pancreaticoduodenal and middle colic veins forms the gastrocolic trunk which empties directly into the superior mesenteric vein at the inferior border of the neck of the pancreas. This provides an exceedingly important landmark for location of the superior mesenteric vein when the pancreas is mobilized to determine operability of pancreatic tumors (see below).

From the surgical standpoint it is important to remember that occasionally the hepatic artery may arise from the superior mesenteric artery and pass behind the pancreas and common bile duct. An unusually long and tortuous hepatic artery may lie close to the superior border of the head of the pancreas or in some cases it may be within the pancreas itself. Under these circumstances, the hepatic artery may be injured during resection of the body and tail of the pancreas and may require ligation. Pierson[6] has pointed out that the distal duodenum and proximal jejunum receive their arterial supply from one of the inferior pancreaticoduodenal arteries, so that interruption of these vessels probably will necessitate resection of the proximal 3 to 5 cm. of jejunum. Anatomy textbooks usually state that the anterior surface of the superior mesenteric and portal veins is free of tributaries in the region of the pancreas. However, the superior pancreaticoduodenal vein often joins the portal vein at the anterior surface of the pancreas, and the gastrocolic vein almost always joins the anterior surface of the superior mesenteric vein inferior to the neck. Other small venous tributaries from the pancreas to the portal and superior mesenteric veins are often present, although hidden from view. The posterior-superior pancreaticoduodenal vein may be the source of severe hemorrhage during mobilization of the portal vein for portacaval shunt.

Lymphatic Drainage

The lymphatic capillaries of the pancreas form richly branched plexuses in the interlobular tissue. These capillaries join to form collecting trunks which empty into the following groups of lymph nodes. The capillaries from the tail drain into the nodes at the hilus of the spleen. From the right side of the pancreas the lymphatics drain into the pancreaticoduodenal lymph nodes lying in the groove between the duodenum and pancreas and into the subpyloric nodes. Anteriorly the drainage is to the superior pancreatic, superior gastric and hepatic nodes along the hepatic artery. Posteriorly the lymphatics pass to the inferior pancreatic, mesocolic, mesenteric and aortic nodes.

Nerve Supply

The sympathetic nerve supply to the pancreas is derived from the greater, lesser and least splanchnic nerves via the celiac ganglia and plexus. These fibers conduct the afferent pain fibers from the pancreas. The sympathetic nerves actually intermingle with branches of the right vagus nerve to form the splenic plexus from which most of the nerve supply to the pancreas emanates. The celiac branch of the right or posterior vagus nerve provides parasympathetic supply to the pancreas. This branch can be spared during vagotomy for duodenal ulcer, as recently proposed by Jackson,[4] Burge[1] and Griffith et al.[3]

SURGICAL APPROACHES AND EXPOSURES

A vertical or a transverse abdominal incision may be used for operations upon the pancreas, depending upon the habitus of the patient and the nature of the surgical procedure. Unexcelled exposure of the entire pancreas is afforded by a bilateral transverse upper abdominal incision if the patient has a wide costal margin, especially when the upper lumbar spine is extended slightly by a firm support. The disadvantage of increased operating time necessitated by such an incision is obviated by the strength of this type of wound. A vertical right rectus paramedian incision gives excellent access to the head of the pancreas, and pancreaticoduodenal resection can be carried out with facility through this incision. Extension into a T-shaped incision may be necessary if total pancreatectomy is desired. The tail and body of the pancreas may be exposed with ease through a left rectus or left Kehr incision.

EXPOSURE AND MOBILIZATION

Perhaps the most common surgical manipulation of the pancreas involves mobilization of the head of the pancreas and the duodenum.

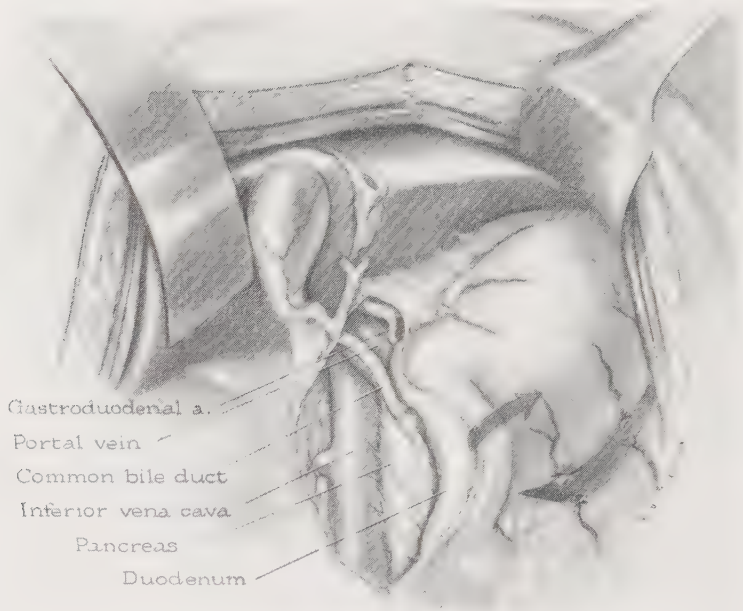

Figure 3. Operative exposure of the head of the pancreas after the Kocher maneuver. Note that portal or superior mesenteric veins are covered posteriorly by the uncinate process, preventing direct vision of these structures inferiorly.

This maneuver is used during exploration of the common bile duct, during gastric resections (especially when a Billroth I reconstruction is anticipated) and for any procedure involving resection or exploration of the head of the pancreas. This technique was first described by Kocher[3] in 1903 and has become one of the most valuable technical maneuvers in the upper abdomen.

The hepatic flexure of the colon is depressed and gentle traction of the duodenum to the left is accomplished by the assistant, thus causing the lateral peritoneal reflection of the duodenum to become taut. The peritoneal reflection can then be easily seen and incised along the entire lateral aspect of the second part of the duodenum. Incision of the loose areolar tissue may be carried to the inferior portion of the hepatoduodenal ligament superiorly and to the point at which the superior mesenteric vessels cross the third portion of the duodenum inferiorly. The duodenum and head of the pancreas may then be gently mobilized to the left to expose the inferior vena cava and occasionally the aorta posteriorly. The portal vein, common bile duct, posterior aspect of the head of the pancreas, and the uncinate process are thus exposed (Fig. 3). The mesenteric vessels are carried anteriorly with the duodenum and pancreas during the mobilization. It is important to point out that the superior mesenteric vessels are covered posteriorly by the uncinate process, a fact often forgotten by many surgeons. Thus, only the suprapancreatic portion of the portal vein is made visible by the Kocher maneuver.

Metastatic invasion of these vascular structures cannot be established with certainty by simple inspection of the posterior aspect of the pancreas after the Kocher maneuver is carried out. If the gastrocolic venous trunk is divided as it enters the superior mesenteric vein at the inferior portion of the neck of the pancreas, the superior mesenteric vessels can be examined more easily for possible involvement by tumor. I have also found that this maneuver affords an excellent delineation of the waist of the pancreas.

Some surgeons advocate insinuation of the finger between the superior mesenteric vein posteriorly and the neck of the pancreas anteriorly, but this cannot be carried out safely until the gastrocolic venous trunk is ligated since severe venous hemorrhage may occur. Moreover, this particular maneuver may tear the posterior-superior pancreaticoduodenal vein at the superior border of the pancreas as well as small venous tributaries from the pancreas. I prefer gentle dissection between the vessels and neck of the pancreas with a blunt-nosed clamp once the gastrocolic trunk has been divided and the posterior-superior pancreaticoduodenal vein has been located. I have not found that ligation of the gastrocolic trunk causes significant impairment of venous return from the colon. When wider exposure of the head of the pancreas is necessary, the hepatic flexure of the colon may be mobilized by dividing its attachments to the posterior parietal peritoneum and retracting it medially.

Gastrocolic Omental Route

This method of exposure of the pancreas provides the widest and most versatile access to the body and tail of the pancreas, as well as a portion of the head. The gastrocolic omentum is placed under slight tension by gentle traction of the stomach superiorly and the colon inferiorly. The gastrocolic omentum is incised and divided between hemostats. This division may be carried as far to the left as is necessary to expose the entire anterior aspect of the neck, body and tail of the pancreas (Fig. 4). The middle colic vessels must be protected during this procedure.

Short segments of the body of the pancreas may then be mobilized by incision of the peritoneal attachment along the inferior border of the pancreas. Portions of the pancreas thus bared may be lifted up and palpated between the thumb and index finger or specimens for biopsy may be removed. Should mobilization of the entire tail and body of the pancreas be necessary, it is accomplished most simply by mobilizing the spleen with the pancreas (Fig. 5). The vasa brevia are ligated and divided. The lienorenal ligament is incised and the splenocolic ligament is divided. The spleen, and the tail and body of the pancreas may be delivered into the wound en bloc after incision of the superior, inferior and posterior thin avascular attachments of the pancreas. This mobilization can be carried out rapidly in an avascular manner as far to the right as the superior mesenteric artery. Resection or mobilization of the body and tail of the pancreas may alternatively be accomplished with the spleen remaining in

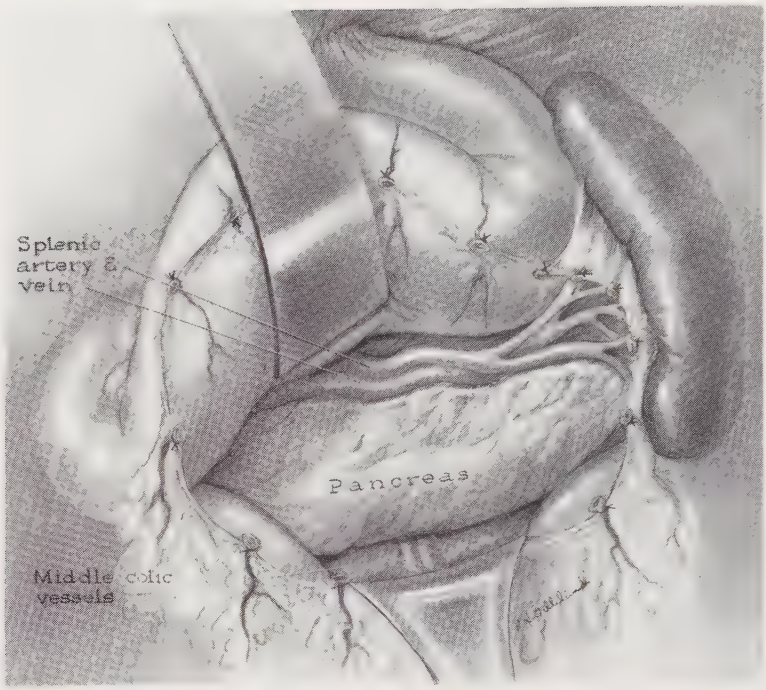

Figure 4. Wide exposure of the pancreas obtained by the gastrocolic omental route. The middle colic vessels must be located and preserved.

situ. This procedure involves incision of the inferior peritoneal attachment of the pancreas, followed by division and ligation of all the branches between the splenic vessels and the pancreas. This method of mobilization is more difficult and time-consuming and is associated with greater blood loss than is mobilization and removal of the spleen with the pancreas.

Exposure through Gastrohepatic Omentum or Transverse Mesocolon

By downward traction of the stomach and upward retraction of the liver, the thin, relatively avascular, gastrohepatic omentum can be exposed and incised. The middle portion of the body of the pancreas is then exposed. Only when the stomach is exceptionally ptotic can more of the pancreas be seen, so that this method of exposure is not generally recommended.

An avascular portion of the transverse mesocolon may be incised to allow entry to the lesser sac after the transverse colon and greater omentum have been elevated in a superior direction. Since care must be taken to avoid the blood supply to the colon, exposure is necessarily limited. Perhaps the prime indication for this route is the presence of a large pseudocyst which has become adherent to the mesocolon and which can be drained into the jejunum through an opening in the mesocolon. A very limited access to the inferior portion of the body of the pancreas can be gained by incision of only the inferior leaf of the transverse mesocolon.

Extraperitoneal Approaches

The posterior aspect of the head of the pancreas may be approached retroperitoneally through a right lumbar incision. The dissection is carried anterior to the right kidney and posterior to the duodenum. The tail of the pancreas may be exposed through a left lumbar extraperitoneal dissection between the colon and the anterior surface of the left kidney.

Both of these methods are time-consuming, complicated, circuitous, and provide only very limited and deep exposure. Only the very occasional retroperitoneal abscess which is strategically located can be drained by this means. Certainly direct access to most of the pancreas is blocked posteriorly by the aorta and vena cava.

SUMMARY

Application of the basic knowledge of the anatomy of the pancreas and its adjacent structures allows meticulous, exacting and avascular dissection of this organ. Thus, complete mobilization and inspection of the pancreas can be achieved by attention to anatomic detail. Exposure of the pancreas is best gained by mobilization of the head of the pancreas (Kocher's maneuver) and by wide opening of the gastrocolic omentum.

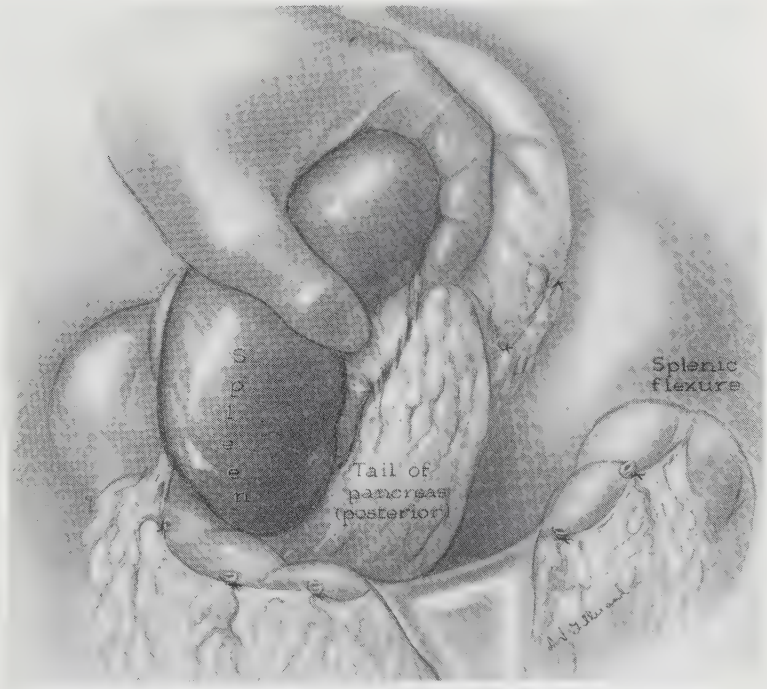

Figure 5. Mobilization of tail and body of pancreas with spleen.

Mobilization of the tail and body are done with the greatest facility when the spleen and pancreas are mobilized simultaneously.

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Surgical Anatomy of the Liver

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Liver surgery during the past decade has developed rapidly after being for years a stronghold of the massive ligature and "cut, pack and run" technique. In place of sporadic case reports and obscure anatomic studies, the surgeon now has available an enlarging clinical and investigative literature to which he may refer when contemplating definitive hepatic surgery. Much of the stimulus for this work is found in the contributions of Lortat-Jacob and Robert,[11] Pack and Baker,[14] Clatworthy, Boles and Kottmeier,[1, 2] and the Quattlebaums,[16] all of whom have accomplished and standardized techniques for the removal of major portions of liver for primary or metastatic malignancy. Correction of the anatomical and clinical findings requires first a consideration of liver embryology.

EMBRYOLOGY

In the beginning, the liver is a ventral bud of the foregut intimately associated with a maze of vitelline and umbilical veins and growing into the ventral mesogastrium from which its capsule and ligaments are ultimately derived. The gallbladder anlage splits away early to an orientation caudad to the main liver mass. By the fifth week of gestation, the liver is found interposed between portal and caval circulations, although temporarily bypassed through the dominant left umbilical vein and ductus venosus, which carry placental blood directly to the inferior vena cava. When the normal bowel hernia is reduced during the tenth week, the liver, which started as a bilaterally symmetrical organ, is challenged for space in the belly by the enlarging stomach. A relative hypoplasia of the lateral portion of the left lobe occurs, and the liver as we encounter it becomes

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the major viscus of the right upper quadrant. At birth the two ends of the venous bypass close, forming the ligamentum teres and the ligamentum venosum. The latter is attached to the left side of the vena cava. The intervening segment remains patent as a portion of the left major branch of the portal vein.

INVESTIGATIVE TECHNIQUES AND FINDINGS

The branching of the portal vein and the other hollow systems determine the segmental divisions of the liver. This anatomy has been investigated chiefly by the use of three techniques:

1. Radiographic studies employing the injection of contrast media either in isolated livers or in the living subject, as in splenoportography.

2. Dye injection studies utilizing differential injection of the right and left divisions of a system with colored dyes to demonstrate segmental or lobar planes.

3. Injection-corrosion studies which fill one or more hollow systems with plastic and then digest away the parenchyma, leaving a cast of the injected systems. The authors have used this last method on 33 livers obtained at autopsy (Fig. 1).

Figure 1. Superior surface of vinyl-acetate injection-corrosion specimen of human liver. Portal vein injected with dark plastic; hepatic veins and segment of vena cava injected with light plastic. Straight line marks plane of falciform ligament. Broken line divides anatomic right and left hepatic lobes. Vena cava at top. Note middle hepatic vein running just to left of broken line indicating lobar division. Main portal trunk seen branching between middle and right hepatic veins. Note prompt division on right into anterior and posterior segmental vessels. The left portal branch runs transversely a considerable distance before bending anteriorly in the umbilical fissure and giving off medial and lateral segmental branches.

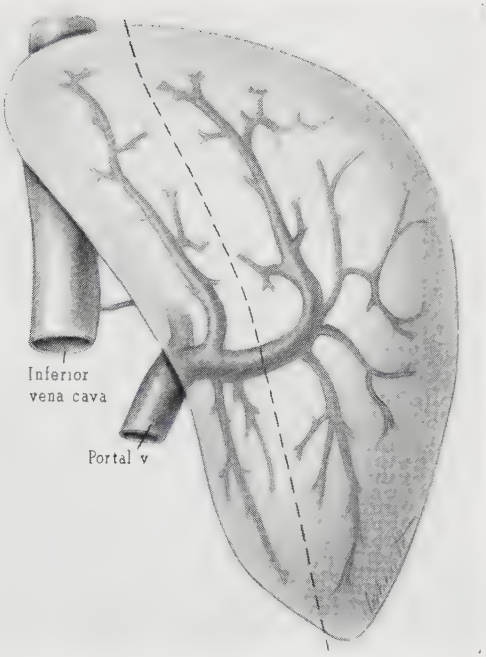

Figure 2. Artist's drawing of right lateral aspect of vinyl-acetate cast shown in Figure 1, depicting the orientation of anterior and posterior segments of the right hepatic lobe.

Intrahepatic Gross Anatomy

Healey, Schroy and Sorenson,[7] Goldsmith and Woodburne,[6] and more recently Hobsley[8] have published exhaustive accounts of such studies on a total of over 200 livers. Although no two investigators are in detailed agreement, all note the same major findings. The portal vein divides deep in the porta hepatis to form a "T," the left main branch being consistently longer and the right varying in length from 3 cm. to zero, depending on the level at which major segmental branches originate. The extrahepatic anomalies of the bile ducts and hepatic arteries are well known. Of particular significance is the frequent origin of a right hepatic artery from the superior mesenteric trunk. Once within the liver substance, however, the hepatic arteries and bile ducts follow the portal vein branches in a fairly constant distribution. On the right there are anterior and posterior liver segments with an oblique line of division. The anterior segment is more accessible and encompasses more of the superior surface of the liver (Fig. 2). On the left, the main portal branch runs laterally at first and then curves anteriorly nearly at a right angle to line up with the umbilical fissure. This represents the only remaining patent segment of the placentocaval bypass of fetal life. It occupies the plane of division between the major medial and lateral segments of the left lobe.

Herein lies the discrepancy between the older concept of the right and left lobes and the actual intrahepatic lobar arrangement. As Figure 1 demonstrates, that part of the liver which is to the left of the falciform

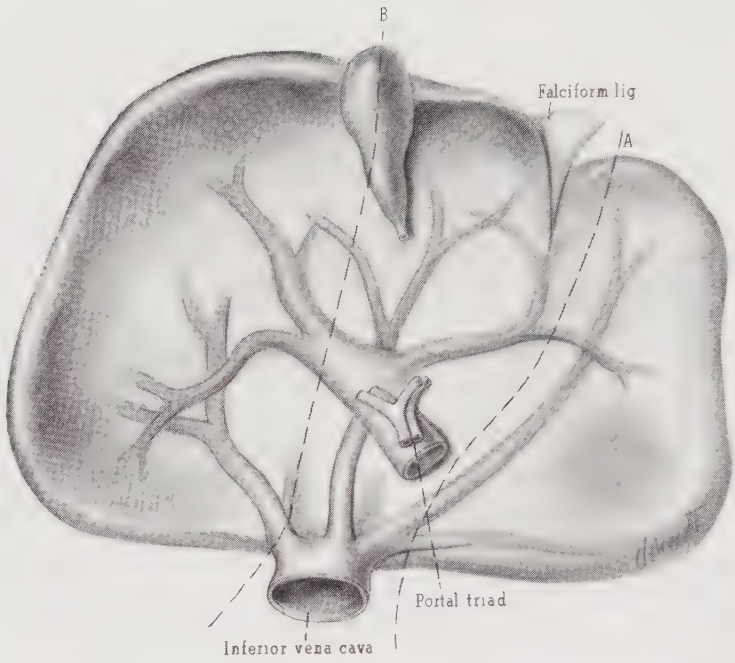

Figure 3. Schematic drawing of intrahepatic arrangement of main vessels related to inferior surface of liver. Note placement of line for left lateral segmental resection just to the left of the umbilical fissure and of the anterior division of left main portal vein. Line for ideal lobar resection is similarly placed to avoid middle hepatic vein.

and round ligaments as they correspond with the umbilical fissure (solid line) is actually only the lateral segment of the left lobe. The so-called quadrate lobe is the medial segment of the left lobe. The caudate lobe receives contributions from both the right and left portal triads. Thus the interlobar plane of division runs from the gallbladder fossa back to the middle of the inferior vena cava as demonstrated by the broken line in Figure 1. Further subdivisions of these major segments of each lobe have been described anatomically, but they have no clinical significance.

Hepatic venous drainage also shows a fundamental bilaterality. Although right, left and middle trunks are fairly constant, the middle hepatic vein frequently empties into the left hepatic vein, or drains by a common ostium with it. The two major divisions on the right side have a somewhat comparable arrangement. The right main hepatic vein proper is often as wide as it is long. The salient feature when resection is contemplated is the lack of congruence of the portal and hepatic distributions. Throughout much of the liver they course at right angles to one another. Figure 1 shows the middle hepatic vein running directly in the plane of division of the right and left lobe as it drains parts of the anterior segment of the right lobe and the medial segment of the left lobe. There are, in short, no

white lines or bloodless planes in the liver. Any plane selected for excision is a compromise, be it for a total lobectomy or a left lateral segmental excision. The transhepatic incision must be planned in a manner calculated to leave as little parenchyma as possible bereft of its nourishment or drainage (Fig. 3).

Lending urgency to this consideration is the finding that each of the four hollow systems is an aborization but in no sense a network. "Accessory" arteries or ducts, in the usual sense of a duplication or collateral supply, do not exist. Irregular vessels, better termed "aberrant" or "replacing," are found to supply definite lobes, segments or areas, but they do not anastomose. Essentially every artery is an end-artery, every vein an end-vein, and there are no anastomoses between bile radicles. There appear to be no significant macroscopic cross connections in the normal liver.

SURGICAL APPLICATIONS

Clinical reports and deductions have often been at variance with these anatomic observations. For example, it is readily demonstrable on certain livers that, if one follows blindly the technique initially described for right lobectomy of transecting liver substance at the falciform ligament, the remaining left hepatic lobe will be entirely without afferent blood supply or biliary drainage. In other subjects, due to the variation in the site at which the left portal triad enters the liver substance, this plane is quite feasible, although the middle hepatic vein is in jeopardy posteriorly, as is the anterior bend of the main left portal branch in the umbilical fissure. It is of interest in this connection to note in published case reports occasional reference to cyanosis of the medial portion of the remaining left segment and in others to postoperative drainage of necrotic liver substance.

Final conclusions as to the planes of surgical excision are naturally dictated by the location and extent of the lesion present. However, given a right-sided lesion adequately clear of the anatomic plane from gallbladder fossa to vena cava midline, this is the plane of choice for right lobectomy. When the procedure is done for gallbladder carcinoma, the majority of the quadrate lobe must be included, with the middle hepatic vein and anterior bend of the left portal vein in mind if not in view. For left segmental excision, the plane a centimeter or so to the left of the umbilical fissure is desirable for the same reason. Frequently the surgeon is guided in the selection of these vital planes of division by color changes in the liver substance secondary to the preliminary ligation of the blood supply.

Technique of Right Hepatic Lobectomy

The usual sequence in right lobectomy begins with exposure through a generous thoraco-abdominal incision. The diaphragm may be incised peripherally or from the eighth interspace to within 2 cm. of the vena

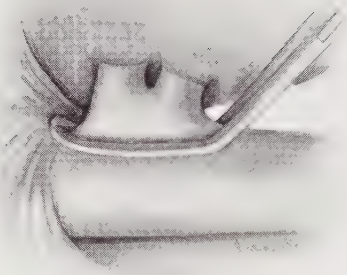

Figure 4. Illustration of technique which safeguards against hemorrhage and air embolism and maintains caval flow while the large-caliber right hepatic vein is controlled.

cava to give adequate room for displacing the liver into the chest and still not harm any major branches of the phrenic nerve (Merendino, Johnson, Skinner and Maguire[13]). The right triangular and coronary ligaments are incised to facilitate mobility, and precautionary tapes are placed around the inferior vena cava just below the diaphragm and distal to the the last hepatic vein. In addition to the major hepatic veins already described, there will be found two to twelve minor hepatic veins. Those minor veins which supply the right lobe are divided between ligatures in order to avoid tearing them while the porta hepatis is dissected. Care must also be taken that the cava is not occluded too long by manipulation of a large liver to expose its porta.

The common hepatic duct, hepatic artery and portal vein are usually accessible in that order, and branch progressively higher in the porta. Trifurcation of the portal vein occurs occasionally and must be kept in mind. In this circumstance the right anterior and posterior segmental trunks are given off directly from the portal vein itself without formation of a right common trunk.

The major complications of lobar resection have been recognized by most authors as air embolism, hemorrhage and bile leakage. It is when the dissection of the right major hepatic vein is begun that the first two of these become imminent. An intrahepatic inferior vena cava may at times necessitate the division of the right hepatic vein within the liver substance. However, even without this troublesome anomaly, the proximity of the right to the middle hepatic vein and the dimensions of the right hepatic vein usually make the attempted control of this vessel by ligature entirely impractical. A Satinsky or other suitable vascular clamp applied to isolate the venous stoma and maintain caval flow (Fig. 4) usually allows enough clearance to ligate the hepatic end of the vein or to apply a second similar clamp. Both stoma and proximal vein are then closed with arterial silk. Blood loss is avoided and air is kept out of the cava.

Actual division of the liver parenchyma goes surprisingly well if, after incision of Glisson's capsule, the scalpel handle is used for blunt

dissection until a hepatic trinity or vein is encountered. These are then divided between clamps. Points of bleeding and bile leakage are readily controlled by stick ties. A T-tube in the common duct minimizes postoperative bile leakage from the cut surface, and may also serve to demonstrate larger open bile radicles at operation by injection of the tube with methylene blue or sterile milk with the distal common duct occluded.

Biliary Drainage

Left segmental excision has been advocated by Peterson and Krippaehne[15] as a step in autograde probing of a bile radicle in order to approach more safely the dissection of a scarred porta hepatis for major bile duct repair. It should be remembered, however, that if scarring has gone above the bifurcation of the common hepatic duct, one may be able to locate only the left major branch by this maneuver.

Similarly the Longmire procedure of intra-hepatic cholangiojejunostomy may drain the left lobe but will do nothing about the right lobe with a totally obstructed right hepatic duct. In a subsequent communication, Longmire and Lippman[10] have emphasized the desirability of operative cholangiography in order to establish the extent of drainage to be anticipated from the procedure.

Diefendorf[3] has used bilateral hepatojejunostomy for biliary decompression above a duct tumor at the porta hepatis to afford the patient 26 months of palliation.

ABERRANT BILE DUCTS

The surgical implications of extrahepatic aberrant bile ducts have been adequately treated in the literature, a portion of which deals with the concept of the "hepatocholecystic duct." Ducts between the liver and the gallbladder fundus are found in some lower animals, but the embryology of the human gallbladder, whose anlage separates early from that of the liver, provides scant basis for a normal liver-gallbladder duct. Anatomic studies by Flint,[4] Healey et al.[7] and Lurje[12] failed to demonstrate such a connection, although in an appreciable percentage of cases they did find sizable ducts in the gallbladder bed. Critical review of published accounts of supposed hepatocholecystic ducts found at operation suggests strongly that these are either postinflammatory artefacts or normal bile ducts which may be found in the gallbladder fossa in one-third of cases. A recent case encountered by one of the authors (J.H.F.)[5] illustrated this point. Two large ducts were transected in the gallbladder bed outside of liver substance which appeared to connect with the gallbladder. However, careful dissection freed them from the gallbladder wall with no demonstrable connection, and the gallbladder in fact contained "white bile" in contrast to the golden brown bile issuing from the ducts.

Figure 5. Photograph of the left triangular ligament from an autopsy specimen. The nodularity of the liver tissue is secondary to cirrhosis. The clamps are on the diaphragm. The common bile duct has been injected with vinyl acetate at the liver hilum, and the arrow points to a plastic-filled biliary radicle extending to the diaphragm. This specimen also illustrates the gradual thinning out of hepatic tissue characteristic of this area.

Aberrant subserosal ducts have also been noted from time to time, most prominently on the anterior surface of the liver, suggesting their possible use in biliary decompression.

However, of more pressing clinical importance are the grossly recognizable bile ducts in the left triangular ligament (Fig. 5). Rapant and Hromada[17] first documented these in the surgical literature and reported two cases in which bile leakage followed division of the ligament in approaching the esophageal hiatus. One of us (E.E.W.)[5] has had one case in which this was suspected but not proved. Reports on the incidence of this anomaly are not available. However, considering the frequency with which the left triangular ligament is divided and the left lateral segment of the liver turned down, demonstration of large bile ducts in this area in three of our first ten injection-corrosion specimens[5] has been sobering.

The explanation of this occurrence is based on the relative atrophy embryologically of this portion of the left lobe, as indicated by the ligament's anatomic name, *appendix fibrosa hepatis*. The reason transection of the ligament does not result in more postoperative bile accumulations within the abdomen very likely has to do with the tension under which the ligament is placed at the time of division, so that parietal diaphragmatic peritoneum rather than ligament per se is what is actually incised. Nevertheless, the possible presence of patent bile ducts in this allegedly avascular and otherwise nondescript structure merits consideration prior to abdominal closure.

SUMMARY

1. Attention to intrahepatic anatomy, specifically related to the distribution of vascular and biliary systems, allows of logically conceived and technically feasible ablation of lobes or segments of the liver for a variety of conditions.

2. Biliary drainage procedures to be effective must take account of the fundamentally branching, nonanastomosing distribution of the biliary duct system at lobar, segmental and subsegmental levels.

3. Aberrant bile ducts encountered at times in the gallbladder bed, on the superior surface of the liver subserosally, and in the left triangular ligament may at any time become of urgent surgical importance to one working in these areas.

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Embryology and Surgical Anatomy of the Extrahepatic Biliary Tract

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The extrahepatic biliary tract probably has more anomalous structures and relationships than any other area of the body. This is true not only in the extrahepatic biliary tract itself but also in its vascular supply. The young surgeon is frequently dismayed and confused by the high incidence of aberrant structures and their anomalous relationships. This review of the embryology and surgical anatomy of the biliary tract was undertaken to enable him to recognize the significance of the anomalies he may encounter. Thus he will be prepared to carry out the meticulous, well planned, widespread anatomic dissection necessary for proper visualization and treatment of diseases of the extrahepatic biliary tree.

EMBRYOLOGY OF THE EXTRAHEPATIC BILIARY SYSTEM

When it is about 3 mm. long (fifth week of intrauterine life), the human embryo has a thickened area of entoderm on the ventral surface of the primitive gastrointestinal tract just distal to the junction of the foregut with the midgut. This small diverticulum is the anlage for the development of the liver, extrahepatic biliary ducts, gallbladder, and the ventral bud of the pancreas. As the hepatic diverticulum grows out into the ventral mesogastrium, it sacculates and divides into superior and inferior caudal buds. From the superior surface of the diverticulum proximal to the enlarging terminal sacculations, the ventral pancreatic bud appears (Fig. 1). The cranial sacculation of the diverticulum (the larger of

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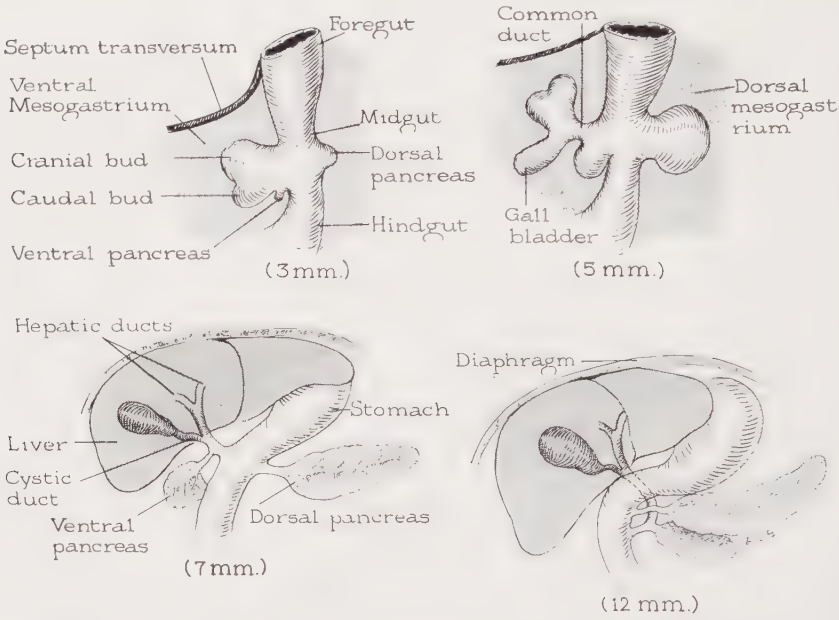

Figure 1. Development of extrahepatic biliary tract in the embryo from the 3-mm. to the 12-mm. stage.

the two terminal sacculations) pushes its way ventrally and superiorly into the primitive septum transversum which separates the thoracic from the celomic cavity. This cephalic bud is composed of a solid mass of entodermal cells spreading out within the substance of the septum transversum as right and left solid cellular columns, which eventually form the right and left lobes of the liver. The growth superiorly of the cranial portion of the hepatic diverticulum, stretching from the duodenum to the liver, is the mechanism for the formation of the hepatic, common hepatic and common bile ducts. The caudal portion of the initial terminal sacculation of the primitive hepatic diverticulum develops into the gallbladder and the cystic duct. It should be noted that the sacculation responsible for the primitive gallbladder and cystic duct is carried away from the primitive duodenum by the elongation of the cranial portion of the same diverticulum.

When the embryo is 5 mm. long the lobes of the liver are forming, and a solid cylinder of entodermal cells represents the extrahepatic biliary tract with its gallbladder and cystic duct offshoot.

At the beginning of the seventh week of intrauterine life, vacuolization takes place within the solid entodermal cellular mass of the extrahepatic biliary tree. A lumen then forms within the elongating common bile duct, hepatic and cystic ducts and gallbladder.

Before the embryo grows to a length of about 7 mm. the common bile duct is attached to the ventral aspect of the duodenum in close contact with the ventral pancreatic bud. As the fetus reaches the 7-mm. stage,

concurrent rotation of both the duodenum and the ventral pancreatic bud occurs, accompanied by the proximal portion of the extrahepatic biliary tract. After the left-to-right rotation is completed, the entrance of the common bile duct into the left posterior surface of the duodenum is seen.

EMBRYOLOGIC ERRORS RESULTING IN CONGENITAL MALFORMATIONS OF THE EXTRAHEPATIC BILIARY TRACT

Abnormalities of the Gallbladder (Table 1)

Rarely an additional bud may be present at the site of junction of the hepatic, cystic and common bile ducts. Failure of this bud to regress results in a double gallbladder. Anomalous positions of the gallbladder are probably due to improper migration of the original gallbladder diverticulum, or to the development of a secondary caudal diverticulum with coincidental failure of the normal diverticulum to develop. A septum of the gallbladder, aberrant valves of Heister, diverticula and congenital stricture are the result of incomplete resolution during vacuolization of the solid cord stage. Congenital absence of the gallbladder is due to failure of the caudal portion of the original hepatic sacculation to develop or lack of proper vacuolization during the 5-mm. stage when the caudal diverticulum is in the solid state.

Other Anomalies of the Extrahepatic Biliary System

1. *Atresia* or *stenosis* of any portion of the extrahepatic biliary tract

Table 1. *Classification of Abnormalities of the Gallbladder*

1. Anomalous forms
   a. Double gallbladder
      (1) "Y" type
      (2) "H" or ductilar type
      (3) Trabecular type
   b. Bilobed gallbladder
   c. Hourglass gallbladder
2. Diverticulum of the gallbladder
3. Congenital absence of the gallbladder
4. Abnormal position of the gallbladder (it is important to remember that no matter where the gallbladder is located, the cystic duct always crosses to the right side of the abdomen and joins the common duct in a comparatively normal relationship)
   a. Abdominal wall
   b. Falciform ligament of the liver
   c. Intrahepatic gallbladder
   d. Retroperitoneal gallbladder
   e. Left-sided gallbladder
5. Abnormalities of the mesentery of the gallbladder (absent, too short or too long)
6. Miscellaneous abnormalities
   a. Aberrant gastrointestinal mucosa in the gallbladder wall
   b. Aberrant pancreatic tissue in the gallbladder wall

results from lack of complete vacuolization of the solid intraluminal mass of the 5-mm. embryo. Any segment of the extrahepatic biliary tract, ranging from the right and left hepatic ducts to the terminal portion of the common duct, may be completely obliterated or stenotic. Multiple combinations of atresia or stenosis in varying areas of the extrahepatic biliary tree are not uncommon. In the newborn this anomaly, which occurs with equal frequency in the two sexes, results in jaundice.

2. *Accessory extrahepatic biliary ducts* result from two or more buds being given off from the primitive biliary anlage. Canalization of these extra buds leads to the formation of accessory ducts. Usually these accessory buds disappear. If they persist in the cephalic portion of the extrahepatic biliary tree they remain as an accessory bile duct or ducts, most commonly running from the liver into the gallbladder. However, they may run from the liver directly into an hepatic, common bile or cystic duct. In biliary tract surgery such a duct, particularly if small, may be unknowingly divided. This is a prime argument for routine drainage of the right upper quadrant after cholecystectomy. An accessory duct in the proximal portion of the biliary anlage may result in a diverticulum of the second portion of the duodenum which protrudes from its posterior medial aspect near the opening of the common bile duct into the duodenum.

3. *Cystic disease of the common duct*, a rare condition, occurs nine times as often in girls as in boys. A large supraduodenal cyst forms between the inferior layers of the lesser omentum. The hepatic and cystic ducts open into the cyst and the common bile duct empties it. The flow of bile into the duodenum is never completely obstructed. The condition may be due to a valvelike obstruction of the common bile duct at the junction of the supraduodenal and retroduodenal portions, resulting from an abnormal peritoneal fixation of the first and second portions of the duodenum.

SURGICAL ANATOMY OF THE EXTRAHEPATIC BILIARY SYSTEM

Intrahepatic and Extrahepatic Ducts

The two main hepatic ducts (right and left), start the delivery of bile from the liver into the gastrointestinal tract. As a rule the right and left hepatic ducts are formed within the substance of the respective hepatic lobes. It is not unusual for as much as one-half to three-quarters of an inch of the major duct to be actually within the hepatic tissue. This anatomic fact is of considerable importance in procedures in which an additional length of the biliary duct is needed for plastic repair or anastomosis. Careful, sharp dissection within the hepatic tissue will usually quite readily bring the ducts into view. Generally, the intrahepatic portion of the left hepatic duct is easier to delineate and its tributary branches join it more deeply within the liver than is the case with the right hepatic duct. Usually, the hepatic ducts join just inferior to the porta hepatis to form the common

hepatic duct. However, the right and left hepatic ducts may continue caudally as separate ducts down to the level of the junction of the cystic duct with the common biliary duct. They then join at this level to form the common biliary duct along with the cystic duct. Occasionally, the hepatic ducts may not join until after the cystic duct has joined the right hepatic duct. The common hepatic duct usually formed by the junction of the right and left hepatic ducts varies in length from 1 to 2.5 cm. Like the hepatic ducts, it lies between the ventral and dorsal layers of the superior portion of the hepatoduodenal ligament. Traction inferiorly upon the superior portion of the duodenum, the pylorus of the stomach and the hepatoduodenal ligament itself usually will bring the hepatic ducts and the common hepatic duct into the operative field.

As the hepatic ducts converge from the hilum of the liver they are separated from the adjacent right and left hepatic arteries and veins by areolar tissue. This tissue normally sheaths all the structures within the hepatoduodenal ligament. The condensation of fibrous tissue about the hepatic ductal system begins with the smaller of the terminal biliary radicles within the liver and then accompanies the right and left hepatic ducts as they emerge from the liver. Consequently, the area of junction of these ducts is partially obscured by a fibrous sheath which extends inferiorly down onto the common hepatic duct to be finally fused with its outermost layer, an important but often neglected feature of dissection (Fig. 2). Because of this strong ventral sheath of fibrous tissue, which binds the hepatic ducts to the normally adjacent, dorsally placed hepatic vessels, the relationship of the upper end of the common hepatic duct to the hilum of

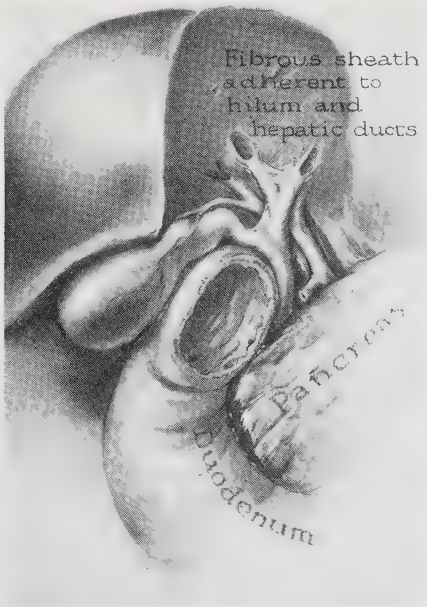

Figure 2. Note the fibrous sheath surrounding the junction of the hepatic ducts with the common bile duct extending both superiorly into the liver and inferiorly onto the common bile duct.

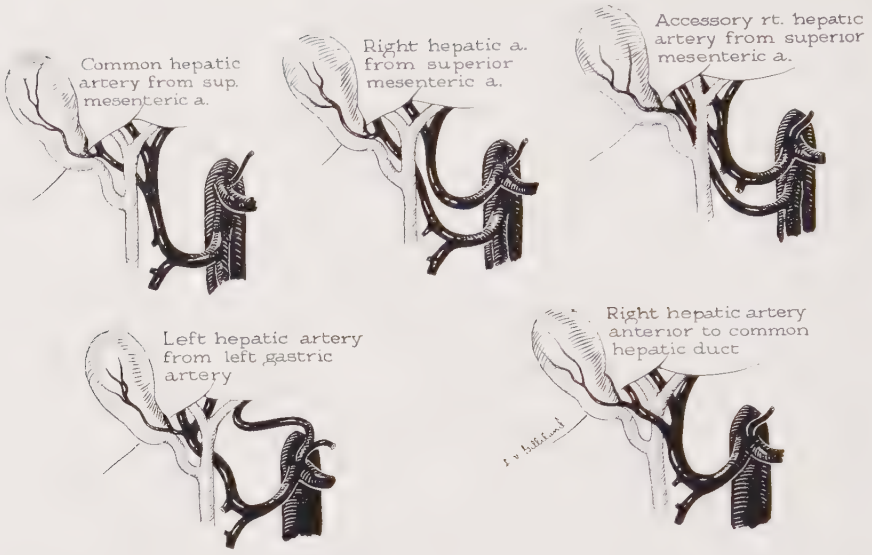
Common hepatic artery from sup. mesenteric a.
Right hepatic a. from superior mesenteric a.
Accessory rt. hepatic artery from superior mesenteric a.
Left hepatic artery from left gastric artery
Right hepatic artery anterior to common hepatic duct

Figure 3. Variations in the relationship of the right hepatic artery to the right hepatic duct and common hepatic duct.

the liver is not easily altered. As a result, previous trauma or inflammation should have little effect upon the location of the ducts or portions thereof near the hilum of the liver.

The relationship of the right hepatic and common hepatic ducts to the right hepatic artery is extremely variable. The artery most commonly crosses from left to right behind the right hepatic duct just above its junction with the left hepatic duct to form the common hepatic duct. However, it may pass ventral to the duct, it may cross the right hepatic duct ventrally or dorsally at a considerably lower level, almost paralleling the cystic artery, or it may not cross the right hepatic duct until just before the duct enters the liver (Fig. 3).

Accessory hepatic ducts (present in 2 to 5 per cent of the population) usually represent merely an extrahepatic union of ducts which ordinarily should join within the substance of the liver. When present they are usually quite short and may run from either lobe of the liver into the subjacent right or left hepatic ducts. Occasionally, they may run directly from the right lobe of the liver to and through the wall of the gallbladder into the lumen. These ducts are not to be confused with small bile canaliculi which also may drain directly from the liver into the adjacent gallbladder.

Gallbladder

General Anatomy. The gallbladder, a pear-shaped organ, can for purposes of general description be divided into four parts: fundus, body, infundibulum and neck. The organ lies on the undersurface of the right lobe of the liver with its neck pointing ventrally, medially and inferiorly.

Generally, it is completely covered by peritoneum over its entire free surface; the reflection of the peritoneum onto the undersurface of the liver helps to attach it to the liver. The deep surface not covered by peritoneum is attached to the liver by extremely vascular, loose areolar tissue. Occasionally, the gallbladder may be completely covered by peritoneum and is attached to the undersurface of the liver by a mesentery of varying length and mobility.

The fundus of the gallbladder usually projects about 1 cm. beyond the mesial portion of the free edge of the right lobe of the liver. Occasionally, the fundus is kinked, forming the so-called Phrygian-cap anomaly.

The fundal tip lies in the angle between the right costal and the right lateral borders of the rectus abdominis muscle in close contact with the anterior parietal peritoneum. This accounts for the ease with which a distended gallbladder is palpated and the early signs of peritoneal irritation resulting from its inflammation.

The posterior surface of the fundus and the entire body of the gallbladder are in intimate relationship with the undersurface of the liver. This relationship is responsible for the spread of gallbladder inflammation directly into the hepatic bed and for the not infrequent rupture of the gallbladder against the undersurface of the right lobe of the liver. The body, infundibulum and neck of the gallbladder lie in close approximation to the first and second portions of the duodenum. In ulceration of the duodenum or in cholecystitis, adhesions between the two organs are frequently formed and occasionally a fistulous tract also may form, permitting passage of gallstones into the gastrointestinal tract.

The body and the infundibulum of the gallbladder are in close relationship to the transverse colon at the point where it crosses the descending duodenum. Adhesions between the transverse colon and the gallbladder are common and biliary stones may occasionally rupture directly into the transverse colon. The serosal-covered surfaces of the gallbladder are closely related to the loops of small bowel which infrequently take up residence in the right upper quadrant of the abdomen.

The infundibulum (Hartmann's pouch) lies between the neck and body of the gallbladder. It presents as a diverticulum lying in close relationship to the undersurface of the cystic duct and frequently hides this structure from view. The infundibulum is attached to the lateral surface of the first portion of the duodenum by a relatively avascular double-layered peritoneal fold derived from the right free border of the hepatoduodenal ligament. This structure is known as the cholecystoduodenal ligament. The first step in the proper identification of biliary tract structures should be the severance of this ligament and concommitant mobilization of Hartmann's pouch. The cystic duct and the site of its junction with the common duct may then be visualized.

The body of the gallbladder continues past the diverticulum known as Hartmann's pouch, tapers into the neck of the gallbladder (which forms

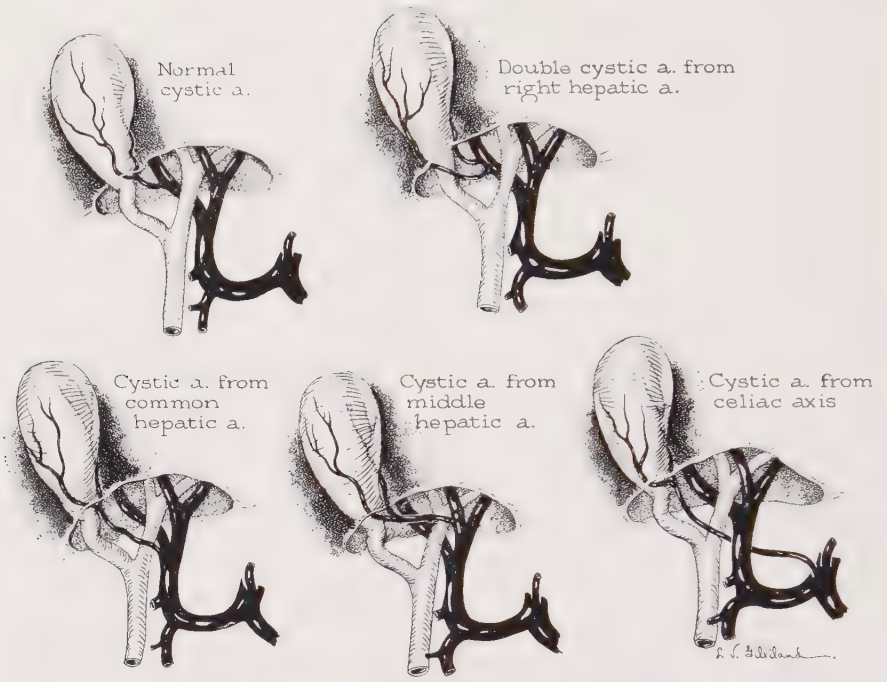
Normal cystic a.
Double cystic a. from right hepatic a.
Cystic a. from common hepatic a.
Cystic a. from middle hepatic a.
Cystic a. from celiac axis

Figure 4. Relationship of the cystic artery or arteries to the cystic duct and common hepatic duct.

an S-shaped curve inferiorly) and terminates in the cystic duct. The neck of the gallbladder is short (5 to 7 mm.) and occupies the deepest portion of the cystic fossa, actually lying in the right lateral free border of the lesser omentum.

The gallbladder and its fossa form a major portion of the upper boundary of the hepatorenal pouch of Morrison, the superior gateway to the broad right lumbar gutter. Postoperative biliary, vascular or purulent drainage may gravitate into the pouch and spread superiorly into the superior-posterior hepatic space or inferiorly from the supramesocolic compartment down the right lumbar gutter to the pelvis.

Blood Supply to the Gallbladder. The cystic artery or arteries usually branch off the right hepatic artery and supply the gallbladder with arterial blood. Most often a single vessel is present which, after branching from the right hepatic artery, runs dorsal to the common duct on its way to the gallbladder (Fig. 4). On reaching the region of the cystic duct, the cystic artery lies superior to that structure in close approximation to it, but it frequently runs inferior, ventral or directly dorsal to the cystic duct. After it leaves the hepatic artery the cystic artery may closely parallel the course of the right hepatic artery for a varying distance, and it may be confused with that structure unless careful complete dissection of the gallbladder fossa is carried out. In 10 to 15 per cent of cases the cystic artery leaves

the right hepatic artery just before that vessel enters the right lobe of the liver. It presents as an extremely short vessel just a few millimeters long which runs directly to the superior portion of the neck of the gallbladder.

In cases of this type the right hepatic artery is often mistaken for the cystic artery. Before either the cystic duct or cystic artery is ligated, a careful, multi-reaffirmed dissection of all the structures of the gallbladder fossa must be carried out. It is a good rule to assume in each case that one or more of the structures in the gallbladder fossa will be anomalous.

Venous Return. There is no cystic vein as such. Venous return from the gallbladder is mainly through multiple small veins which run from the periphery of the gallbladder directly into the hepatic bed. Occasionally, fairly large veins may be closely related to the cystic duct and will follow its course toward the common duct, eventually entering the portal vein.

Lymphatic Drainage. Lymphatic drainage from the gallbladder usually takes one or more of the following routes. It may run directly into the liver through multiple small lymphatic channels, eventually joining with the liver lymphatics. It may drain into a node (cystic node) or group of nodes found overlying the cystic duct just proximal to its junction with the common duct. Drainage from the cystic nodes continues inferiorly down channels in the hepatoduodenal ligament and finally drains into the deep nodal chain about the celiac axis.

Cystic Duct

The cystic duct is short (0.5 to 1.5 cm.), narrow, and runs from the neck of the gallbladder into the common bile duct or rarely into the right hepatic duct. Its mucosa, shaped into spiral folds (valves of Heister), makes it difficult for a stone to pass from the gallbladder into the common duct (Fig. 5). The mode of junction of the cystic duct with the common duct is frequently anomalous. The cystic duct may join the common duct (1) at a right angle, (2) it may parallel the common duct for a varying distance before entering the lateral side of the common duct, (3) it may enter the dorsal surface of the common duct, (4) it may run dorsal to the common duct and then enter it from its left lateral surface, or (5) it may enter the right hepatic duct. Variations of the mode of entrance of the cystic duct into the common duct make extremely careful dissection necessary in order to prevent injury to the common duct at the time the cystic duct is ligated. Care must also be taken not to leave an unrecognized remnant of cystic duct attached to the common duct because of the possibility of late flare-up of inflammation of the stump.

Common Bile Duct

For descriptive purposes the common bile duct is divided into the following areas: supraduodenal, retroduodenal, pancreatic and intraduodenal.

The common bile duct may be from 5 to 10 cm. long although the

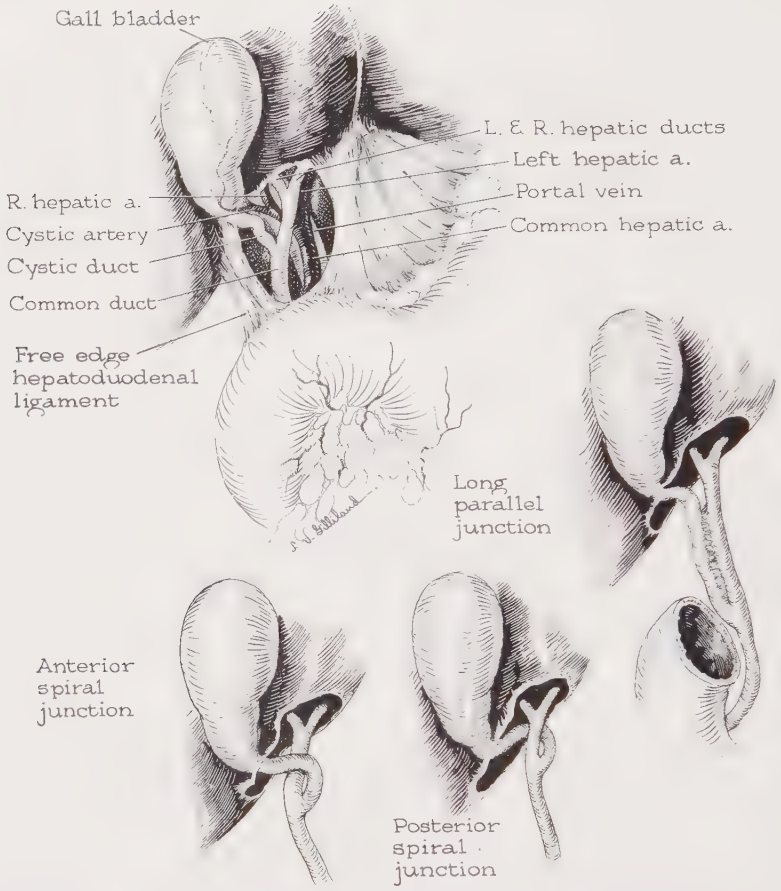

Figure 5. Variations in the length of the cystic duct and its level and mode of entry into the junction of the common hepatic and the common bile duct.

usual length is 8 to 10 cm. It varies in width from 0.38 to 1 cm. but may narrow or widen markedly in biliary tract disease. The ductal walls are composed of mucosa, fibrous tissue and serosa. Only rarely are smooth muscle cells found in the ductal wall.

The supraduodenal portion of the duct is the longest segment (2 to 5 cm.). This is the portion of the duct which is usually inspected for disease and opened for drainage or exploration. It runs in the inferior half of the right free border of the hepatoduodenal ligament between the two serosal surfaces. The hepatic artery lies just to the left of the duct but on the same plane as the duct. The portal vein lies dorsal to the duct and the hepatic artery, separated from them by loose areolar tissue. All three of the aforementioned structures lie ventral to the epiploic foramen just above the superior margin of the first portion of the duodenum. The inferior

vena cava lies dorsal to the common duct, the hepatic artery and the epiploic foramen, and is attached to the retroduodenal tissues.

The retroduodenal portion of the duct, which is 1 to 2 cm. long, lies dorsal to the first portion of the duodenum on its way to the upper border of the pancreas. The descending gastroduodenal artery lies 1 to 2 cm. to the left of the common duct, being closer to it infraduodenally than supraduodenally. This portion of the common duct is frequently involved by the pathologic extension of posterior duodenal ulcer. Inflammation and resultant scarring may draw the duct to the left, subjecting it to possible injury during mobilization of the first portion of the duodenum. An often neglected relationship of the retroduodenal portion of the common duct is its connection with the middle colic artery. This vessel is given off the superior mesenteric artery just inferior to the sweep of the undersurface of the first portion of the duodenum. It lies in a fold of transverse mesocolon which is loosely fused to the inferior surface of the first portion of the duodenum and the pylorus of the stomach. During mobilization of the retroduodenal portion of the common duct this fold must be mobilized and allowed to drop inferiorly.

The pancreatic portion of the common duct, which is 2 to 2.5 cm. long, is related to the dorsal surface of the pancreas in one of two ways. The duct may be entirely retropancreatic or it may be within the substance of the dorsal portion of the pancreatic head. The pancreatic portion of the common duct courses inferiorly and slightly to the left, lying close to the medial surface of the upper portion of the descending duodenum. Here the common duct is closely related to important vascular structures. The descending gastroduodenal artery lies less than 1 cm. to the left of the most superior portion of the pancreatic segment of the common duct. The large pancreatoduodenal vein lying on the dorsal surface of the head of the pancreas is closely related to this portion of the common duct. It is the source of much troublesome bleeding during pancreatoduodenectomy. The Kocher maneuver in the descending duodenum allows for visualization of this portion of the common duct. The pancreatic portion of the common duct terminates on the posterior medial wall of the duodenum about 2.5 cm. below the junction of the first and second portions of the duodenum and just superior to the crossing of the descending duodenum by the transverse mesocolon.

The intraduodenal portion of the duct is the shortest and most anomalous part. The duct runs obliquely through the wall of the duodenum after entering its left posterolateral surface and finally empties into the duodenal lumen via the ampulla of Vater. This portion of the common duct joins with the major pancreatic duct in one of three ways. There may be an extraduodenal junction just external to the duodenal wall or a junction may occur during the tangential course of the duct through the duodenal wall. The common and the major pancreatic ducts may not join

but each may separately enter and discharge on the eminence of the duodenal papilla.

The ampulla of Vater is a reservoir formed by the junction within the duodenal wall of the common duct and the main pancreatic duct. The ampulla opens into the duodenum on its posteromedial surface at the summit of a small elevation called the duodenal papilla. This elevation is frequently hidden from view by overhanging duodenal rugae. The papilla, as well as the ampulla of Vater, is surrounded by a complex sheath of muscle fibers known as the sphincter of Oddi. These fibers are probably continuous with the circular muscle of the duodenum. The ampulla is a favorite spot for the lodging of calculi, and the smooth muscle fibers of the sphincter of Oddi frequently suffer a neuromuscular imbalance. This site of disease may be visualized by mobilization of the second portion of the duodenum by Kocher's technique, followed by duodenotomy.

Arterial Circulation to the Common Duct. The first and second portions of the duct are supplied by small twigs from the hepatic and cystic arteries and the third and fourth portions by the superior pancreaticoduodenal arteries. The great variation in the distribution of arterial vessels to the duct and their unusual anastomotic patterns probably accounts for some of the postoperative ischemic sequelae when long segments of the duct are mobilized and stripped. In this respect the common duct is not too unlike the ureter in the Wertheim procedure.

SUMMARY

The major anatomic structures of the extrahepatic biliary tract have been described. Particular emphasis has been placed on the embryologic growth and development of the extrahepatic biliary tract in order to simplify the clinical problems associated with embryologic errors.

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Anatomical Approach for Completing the Proctectomy Phase of a One-Stage Proctocolectomy for Chronic Ulcerative Colitis

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We have previously presented a technique for completing the final proctectomy phase of coloproctectomy for chronic ulcerative colitis.[1] We herewith present an alternative method applicable in certain exceptional cases. We wish to also suggest a method we believe to be original which serves to reduce the dead space in the presacral area and to deepen the peritonealized pelvis.

The technique originally published and the one we most commonly use is illustrated in Figures 1 to 5. After the patient is anesthetized, the proctoscope is inserted into the rectum and the rectum is completely emptied by irrigation and suction (Fig. 1). In those patients in whom the rectum is diseased to the point where proctectomy is a certainty, the anal sphincter is divided and the anus widely dilated. In all cases a pack 6 inches in length, saturated in benzalkonium chloride (Zephiran), is inserted into the rectum (Fig. 2). It is only rarely that inflammatory strictures make the insertion of this pack impossible. An umbilical tape is attached to this pack. This permits the circulating nurse to withdraw it from below at the proper time in the course of the operation.

The patient is then prepared for laparotomy and a long left para-

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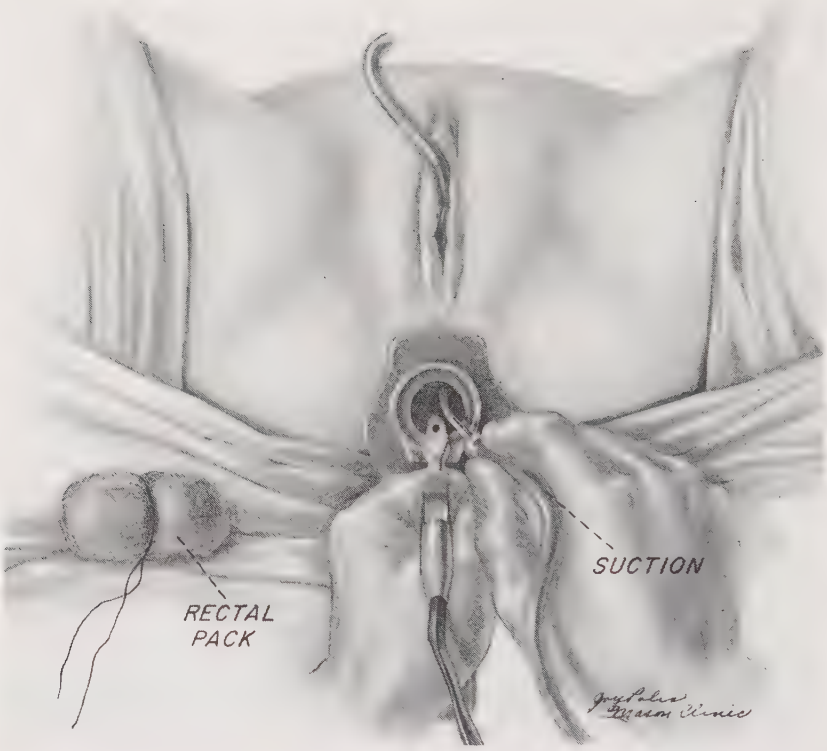

Figure 1. With patient on the operating table a proctoscope may be used to thoroughly empty the rectum by suction. A Zephiran-soaked pack is then placed in the rectum.

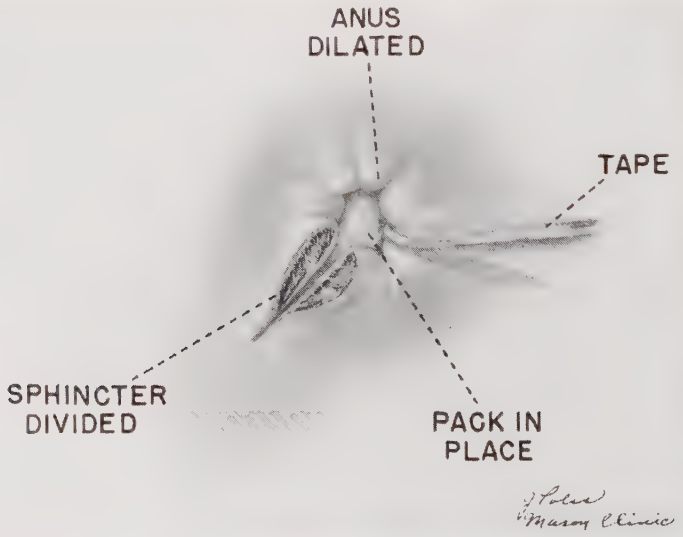

Figure 2. The pack is now in the rectum and the sphincter has been divided.

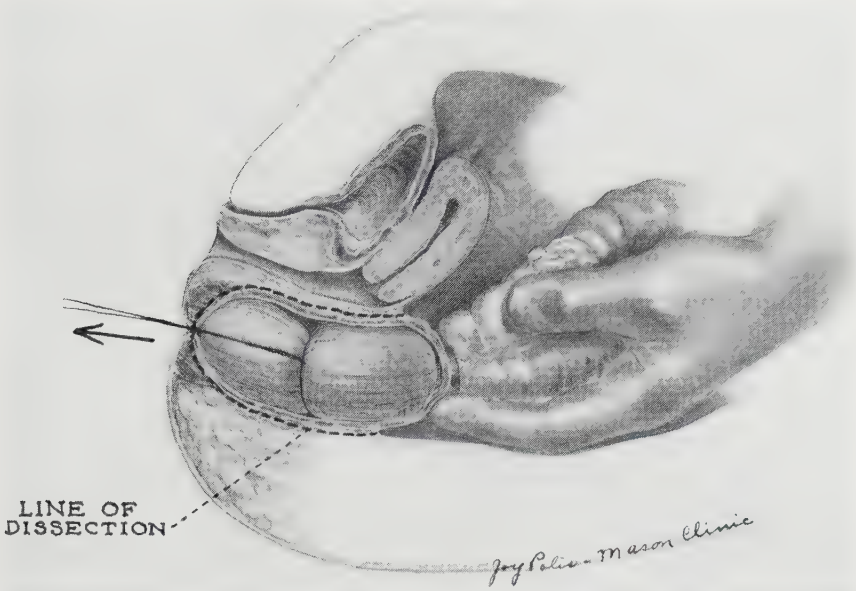

Figure 3. Zephiran-soaked pack in rectum with tape attached for removal.

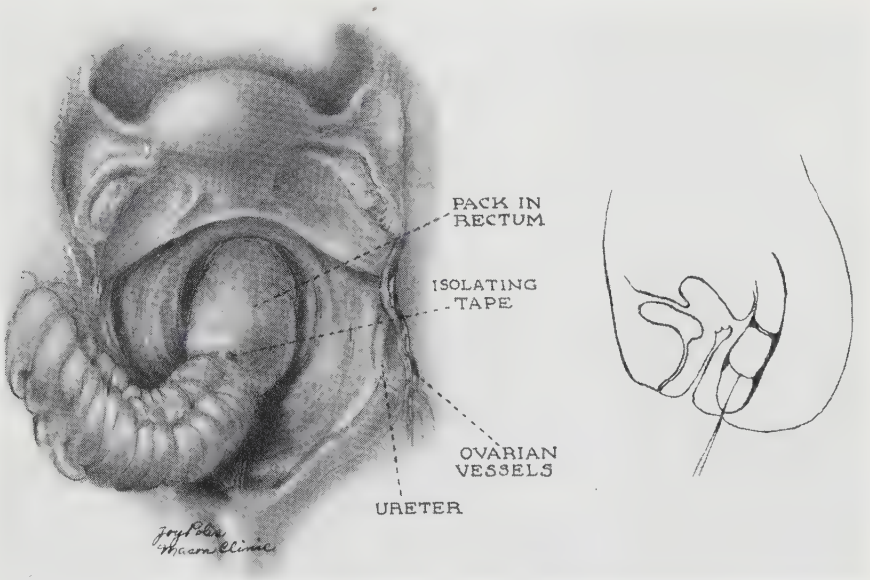

Figure 4. Rectal dissection completed with pack in place.

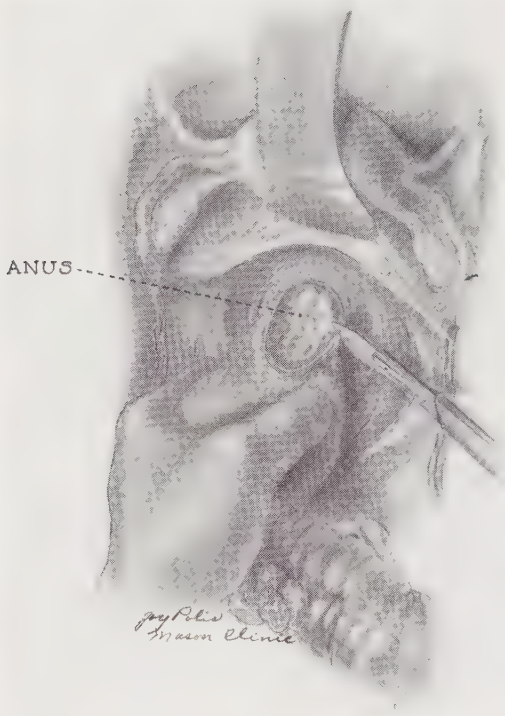

Figure 5. Pack withdrawn. Empty and relatively sterile ampulla opened. Dentate line visualized and rectum incised at dentate line, removing all rectal mucosa.

median incision is made. After thorough exploration, if the plan for proctectomy is to be carried out, a tape is placed about the rectosigmoid junction. This keeps the rectum emptied during the manipulation of the colon. The portrayal of the details of the colectomy and ileostomy is not the purpose of this presentation and for this the reader is referred to the original publication. Suffice it to say that after the abdominal colon has been freed, the rectum is then dissected from its attachments with particular care in the male to preserve as much of the pelvic plexus of nerves as possible and thereby reduce complications of the urinary bladder and risk of impotence. Likewise, since we are not dealing with malignancy, the entire mesenteric investment of peritoneum is freed from the rectosigmoid and is preserved. The importance of this step will be discussed later.

After the dissection is carried down to the pack, a second tape is placed tightly around the bowel just proximal to the pack, thereby further isolating the rectal ampulla from the sigmoid, and thus preventing spillage into the ampulla from above (Fig. 3). The rectal dissection is then completed with the pack in place, down to the levator floor (Fig. 4). This depth is first developed posteriorly, then carried laterally on either side and finally anteriorly to complete the circumferential isolation of the anorectal juncture. Blunt and sharp dissections are used.

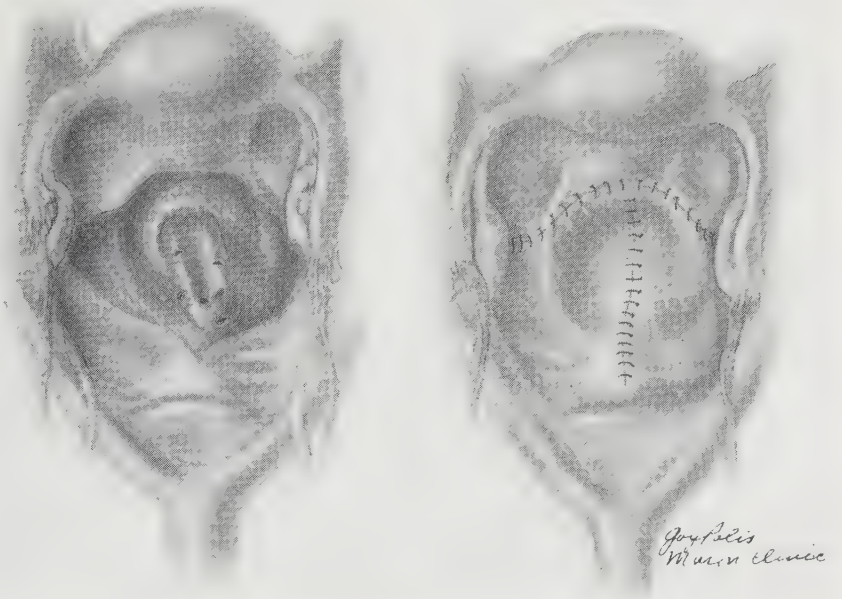

Figure 6. The proctectomy has been completed, a soft rubber sump drain has been placed to emerge through the anus, the pelvic cavity has been peritonealized in a conventional manner.

At this point the pack is withdrawn from below by the circulating nurse. The empty and relatively sterile ampulla can then be opened without fear of spillage into the pelvis. The ampulla is opened close to the anus and the dentate line is visualized. It is of course extremely important to remove all the rectal mucosa. Therefore, the rectum should be removed at, or below, the dentate line (Fig. 5). An incision into the anal skin is preferable to an incision which leaves rectal mucosa behind. A soft sump type of drain is then carried outward through the anal opening (Fig. 6).

A NEW METHOD FOR REPERITONEALIZING THE PELVIS

Whereas in the past we have used the usual T-contour closure (Fig. 6) of the pelvic peritoneum to protect the intestine from gravitating into the bare presacral space, it occurred to one of us (J.W.B.) that this step could be modified to advantage. Instead of the free approximation of the peritoneal edges in a "T" manner across the free pelvis as is done in the more radical cancer resection, the closure is now done as depicted in Figure 7. The approximating stitch is taken in first one edge of the peritoneum, then through the presacral fascia and finally through the opposite edge of

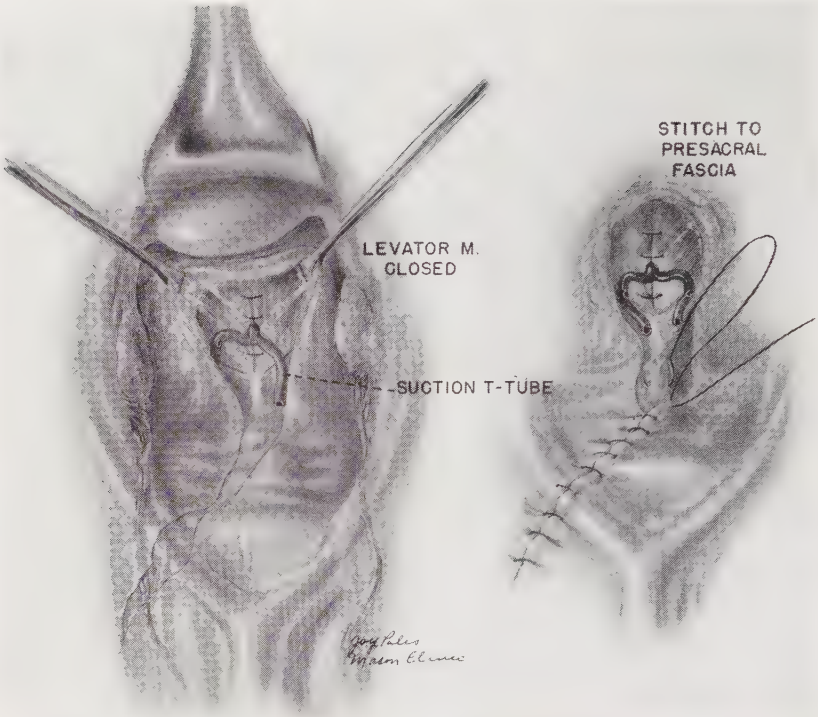

Figure 7. The modified technique for peritonealizing the presacral and pelvic space. The levator muscles have been closed, leaving a soft T-tube latex drain emerging through the anus. The peritoneal edges are approximated by a stitch which also takes a bite in the presacral and levator fascia, thereby obliterating dead space. Branches of the presacral nerve are avoided.

peritoneum. As this stitch is tied, the peritoneal edges are approximated and held down to the depth of the presacral space. These interrupted stitches so placed are continued anteriorly until the presacral space is all but obliterated (Fig. 8). So little space is left that the previously described large sump drain is substituted by a soft 16 French latex T-tube (Fig. 8).

This method has the advantage of obliterating or reducing the dead presacral space. It also eliminates the possible hazard of the intestine rupturing through a free pelvic diaphragm. It has the third advantage of increasing the peritoneal pelvic cavity. It is made possible by the greater salvage of the pelvic peritoneum where radical cancer surgery is not done.

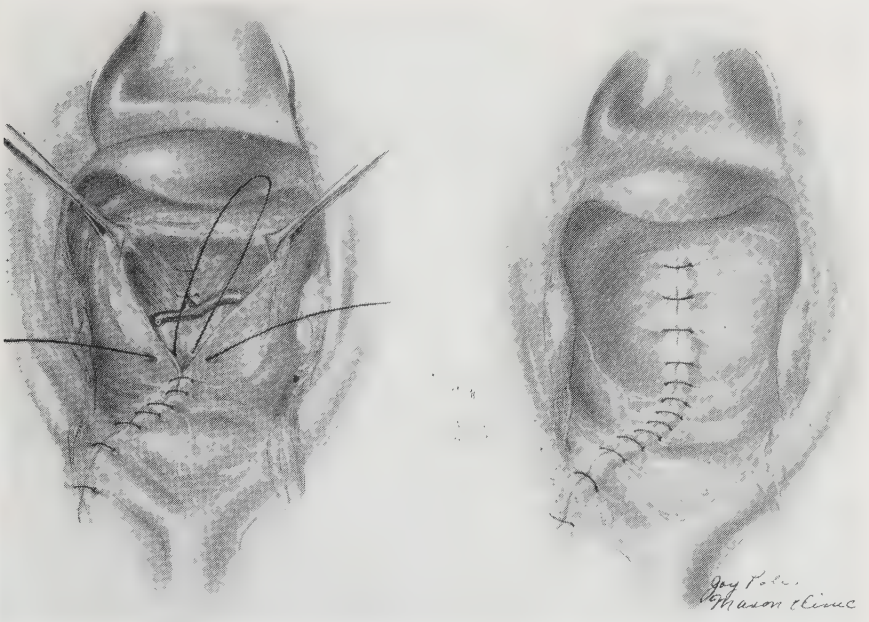

Figure 8. The completion of the peritonealization of the pelvis.

AN ALTERNATE APPROACH TO THE COMPLETION OF PROCTECTOMY

As pointed out in our previous reports, the advantages of the single-stage operation done completely through the abdomen are several. The operation is shortened and turning the patient under anesthesia is avoided. A large perineal wound and scar are avoided as contrasted to the conventional abdominoperineal method. Rehabilitation occurs more promptly.

None of these advantages is sacrificed by modification of this abdominal approach in certain exceptions where the former method is less feasible. We refer to the occasional instance where a very small pelvis, usually male, or where an extensive inflammatory process about the terminal rectum, makes it difficult or, from the standpoint of bleeding, hazardous to complete the dissection all the way to the anus from the abdominal side. Visualization and dissection are more apt to be restricted and therefore more difficult as the dissection is carried anteriorly under the prostate and urethra. If, after the rectum is freed posteriorly down to the levator floor, the dissection forward becomes in the least difficult, we switch the approach temporarily to the perineal side. Because this may in any case become advisable, patients in whom the perineal approach may be necessary are draped to permit this combined approach without repositioning or redraping the patient, just as is done for the Swenson pull-through operation. The advantages of our original approach are, therefore, not lost, while the hazard of stretching

Figure 9. The preoperative draping for the combined approach requires adaptable stirrups which can be moved in multiple planes.

Figure 10. If a possible approach is anticipated, the perineum is draped in a sterile manner, as for the Swenson pull-through procedure.

the limit of the single approach to cover an occasional unusual situation is thereby avoided. The resort to this modification will be less often required by the expert surgeon, but even the most adept will be well advised to use it where bleeding or dissection becomes a problem.

The preoperative draping requires adaptable stirrups which can be moved in multiple planes (Fig. 9). The proctoscopic suction and insertion of pack has been preoperatively accomplished as described above, but in addition the perineum is draped in a sterile manner as for the Swenson pull-through procedure (Fig. 10). The operating table remains broken, a sterile instrument table is positioned to replace the lower half of the operating table, and the draped legs are lowered out of the way during the course of the laparotomy (Fig. 11). It is then no problem to raise the legs by adjusting the stirrups, and by removing the instrument table the surgeon has immediate free access to the perineum at any point in the operation that this may become desirable.

At the time the operation is switched to the perineum the rectal pack can be either removed or the lead tape can be cut and the pack left indwelling. We prefer the latter. The anus is purse-string closed with a suture and repainted with Zephiran. A circumferential incision is made about the anus, just distal to the pectonate line (Fig. 12). An electrosurgical blade is used for ease and to reduce bleeding. A finger is then insinuated between the rectum and sphincter ani circumferentially. The assistant's finger from above is soon felt through the levator diaphragm posteriorly. The abdomen

Figure 11. The distal panel on the operating table is broken downward, and replaced by a draped, sterile instrument table as shown in foreground of this photograph. The draped legs, suspended from the adjustable stirrups, are lowered out of the way during the laparotomy. It will be noted that the legs are horizontal, the humps in the drapes on either side representing the curvature of the stirrup—not the leg itself.

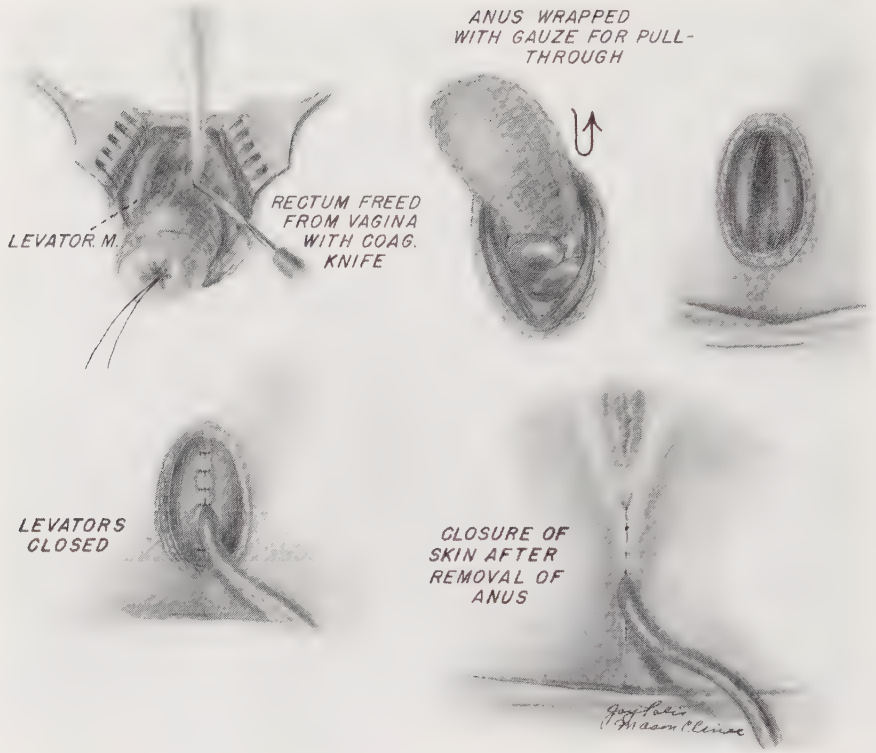

Figure 12. The alternate method for completing the proctectomy phase. See text for details.

is entered at this point and the dissection completed around and anteriorly from the prostate or vagina, as the case may be. A gauze sponge is tied around the end of the anus and the assistant lifts the specimen into the abdominal field. From below the levator muscles are brought together with a few interrupted sutures over a latex T-tube drain. The small perineal incision is approximated by a few interrupted subcutaneous fine chromic sutures about this drain. The legs are lowered, the instrument table is repositioned at the end of the operating table, and the surgeon then returns to the abdominal side. The colectomy is completed, the ileostomy established and the operation thereby concluded.

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Applied Anatomy of the Anal Canal and Rectum

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Most clinicians consider the anal canal and rectum as a single unit, as implied in the terms anorectal disease, anorectal surgery and anorectal canal. The embryologist readily separates the rectum which is derived from the hindgut (entodermal) from the anal canal which develops from the proctodeum (ectodermal). The anatomist finds separation of the two difficult as the anal canal and rectum fuse together, neither beginning nor ending abruptly (Fig. 1). This fusion occurs over a zone several centimeters in width at the level of the levator muscles.[4] As this zone of transition contains both entodermal and ectodermal tissue, various types of neoplasia can occur. This explains why a squamous cell carcinoma, cuboidal ("transitional") cell carcinoma, adenocarcinoma or an adenoacanthoma may be found in the same region.

Note in Figure 1 the greater diameter of the "rectal" ampulla above the levator muscles than below. This marked change in diameter results in folds or columns (columns of Morgagni). The folds end in natural traps, the anal crypts, which face against the fecal stream coming down from above. Seldom seen grossly, but found on microscopic examination, are multiple, tubular, easily occluded ducts that empty into the anal crypts. The anal ducts are at various levels with reference to the soft tissues and muscles that surround the anal canal. The depth and direction of the anal crypts explain the varying penetration and types of fistulas and abscesses that occur, e.g., subcutaneous, ischiorectal and submucosal.

Note, too, in Figure 1 how the region of the columns and crypts is surrounded by the levator and sphincter ani muscles. Contraction, or inability to relax these muscles, deepens the folds and crypts. Tensions (stress) and loose, irritant stools (diarrhea) are definite factors in the development of anal fissures, abscesses and fistulas. Diarrheal stools are

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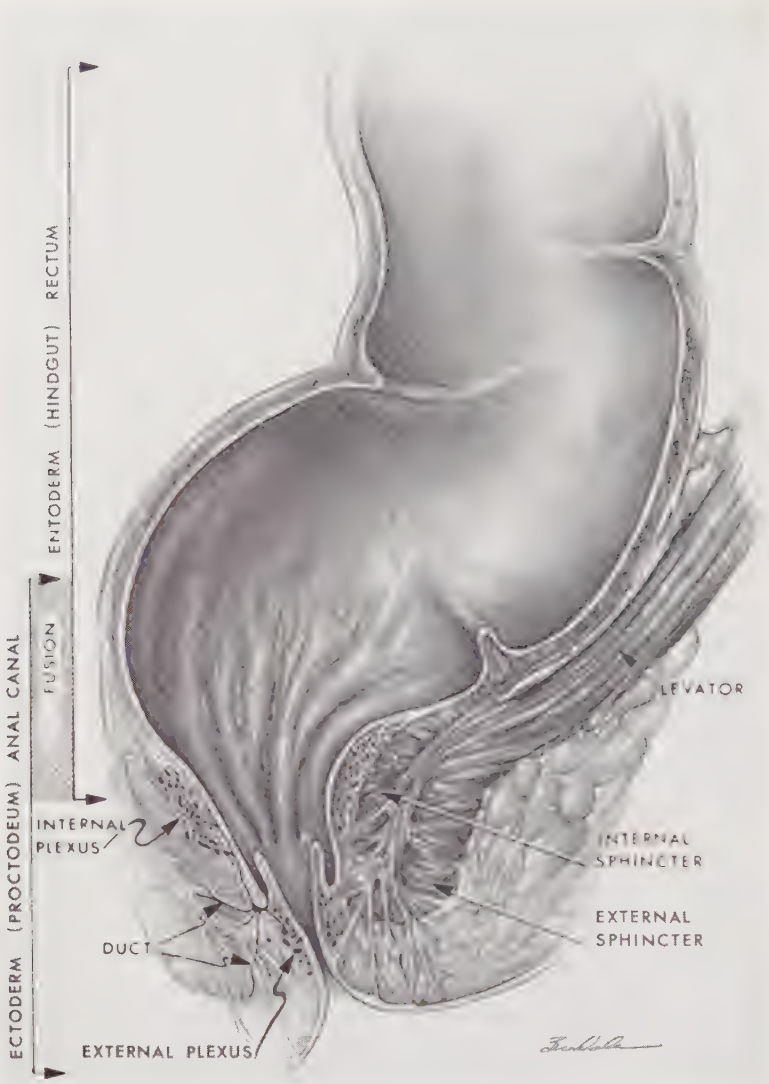

Figure 1. Anal canal and rectum.

chemically irritating, and this irritation causes cellular breakdown of tissue normally resistant to infection. Loose, forceful stools also mechanically gain entry to the anal crypts more readily than formed stools. Enemas and laxatives are mucosal and ganglionic irritants which may cause anal fissures and fistulas, and, when used postoperatively for these conditions, they hinder proper healing. Hydroscopic agents reduce the loose character of diarrheal stools. Used postoperatively, they help to promote formed but soft stools which allow for natural dilatation of the anal canal, which is

much better than mechanical dilatation of the canal with a gloved finger that causes the patient increased pain and apprehension.

Alleviation of the patient's anxiety is important. Tension, anxiety and pain result in muscle spasm. Many patients who have an anal fissure have a history of peptic ulcer or an irritable bowel. Partial sphincterotomy should be done as part of a fissurectomy as well as part of most hemorrhoidectomies.

The anal canal contains multiple folds and crypts, yet the posterior (85 per cent) and the anterior (13 per cent) anal crypts (in the adult) are the usual foci of nonspecific infections (fissure, abscess, fistula). The levator muscle encircles the rectum posteriorly, and portions of the external sphincter muscles are anchored posteriorly (coccyx) and anteriorly (perineum). This results in deeper anal crypts in the midline than laterally. In addition, the hollow of the sacrum, position of the coccyx and angle of the anal canal cause the stool to be directed to the midline crypts. In the *infant* these structures are not well developed. An infant is just as likely to have a lateral fissure or mechanical abrasion of the anal canal as midline involvement. Lateral crypt infection in the *male adult* is frequently due to gonorrhea or syphilis. This type of infection is being seen more frequently in the past ten years. A male adult with a lateral anal fissure or ulcer should have specific etiologic studies including a darkfield examination.

Most patients and many physicians consider all anal skin tags to be due to hemorrhoids. This erroneous concept may result in improper treatment and advice to the patient. Inflammatory anal tags, secondary to crypt infection, differ from hemorrhoidal skin tags in their position and gross appearance.

Figure 2, *A*, illustrates a posterior crypt infection, anal fissure and associated inflammatory anal skin tag and hypertrophied anal papillae. Inflammatory skin tags develop because of edema, round cell infiltration and fibrosis. They are triangular in shape, and, on their inner surface, have a linear sulcus which leads to the infected midline anal crypt. A hypertrophied anal papilla is also secondary to inflammatory change and is not a premalignant lesion.

Advances in studies and surgery for vascular disorders have given us a greater appreciation of vascular arcades, collateral and accessory vessels. The superior, middle and inferior hemorrhoidal arteries by common arcades supply the rectum and anal canal with blood. The termination of these vessels accounts for the presence and location of the larger external and internal hemorrhoidal veins. All adults have hemorrhoidal veins. Hemorrhoidal disease (erosion, protrusion, thrombosis) occurs in three main zones, right posterior, right anterior and left posterolateral, because this is where the hemorrhoidal arterial blood supply terminates (Fig. 2, *B*). Accessory or secondary hemorrhoids occur in the midline of the anorectal canal only after involvement of one or more of the three main hemorrhoidal zones. There are many different techniques of hemorrhoidectomy, and the uni-

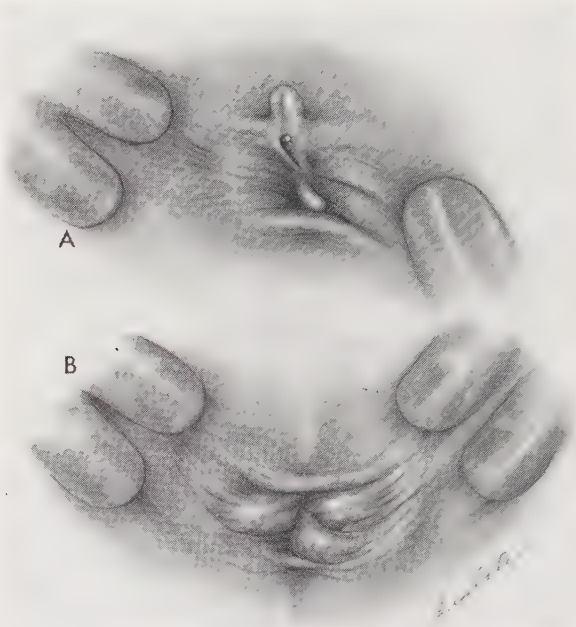

Figure 2. *A*, Anal fissure with associated inflammatory anal skin tag. *B*, External hemorrhoids.

formly successful methods include high and deep ligation at the apex of the internal hemorrhoidal pedicle, where the main arterial supply is present beneath the mucosa in the right posterior, right anterior and left lateral zones.

Figure 3 indicates the difference in nerve supply and sensitivity present above the level of the anal crypts from that below this level. At and below the level of the anal crypts (dentate, pectinate line) the nerve supply is somatic (cerebrospinal) through the pudendal nerve. This area is very sensitive to noxious stimulation such as cutting, pin prick, pressure and heat. However, above the anal crypts the innervation is visceral (autonomic) and comes from the pelvic plexus. Here there is response only to distention or crushing.[5]

This limited response to stimulation above the level of the anal crypts allows for fulguration of polyps and biopsy of lesions without discomfort. Internal hemorrhoids can be injected with an irritant sclerosing agent such as 5 per cent phenol in cottonseed oil, but this is not true of external hemorrhoids which are covered by skin and are below the anus. Hardly a week goes by that a patient or physician does not call and tell of failure to replace a painful thrombosed anal hemorrhoid as if it had "prolapsed" from inside the anus. Manual mishandling of an external hemorrhoid only brings on increased difficulty. Internal hemorrhoids may cause painless protrusion and/or bright red rectal bleeding. Prolapsed internal

hemorrhoids usually respond to manual replacement. Occasionally, both external and internal hemorrhoids will become thrombosed, edematous and painful. The edema is marked and will often give an appearance of "strangulation," but strangulation never occurs because it is impossible for the sphincter to remain contracted because of muscle fatigue. Surgery or "hands off" is the primary treatment for thrombosed hemorrhoids. No attempt should be made to reduce that which cannot be reduced.

Fissures usually cause pain with and after a bowel movement because of their location just below the crypts and over the underlying sphincter muscle. The initial discomfort is "sharp and cutting" in character as the stool passes over the open sore as the canal opens. Following defecation, pain due to a fissure is caused by muscle spasm.

An abscess, in contrast to a thrombosed anal hemorrhoid which is a self-limiting process, brings on increased discomfort as the soft tissue swelling continues. Pain in the "anorectal" area signifies tissue irritation at or below the level of the anal crypts. The character of the pain suggests the cause as a thrombosed *external* hemorrhoid, anal fissure or abscess.

The internal and external sphincter ani muscles, by their very nature,

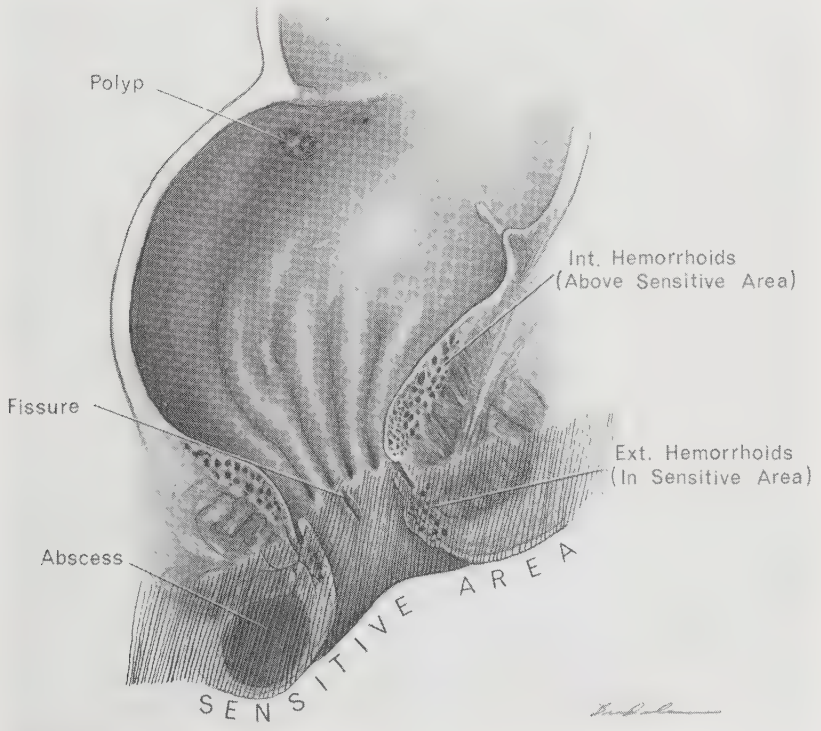

Figure 3. Location of painful conditions in sensitive area of anal canal compared to tissue changes in "nonsensitive" area.

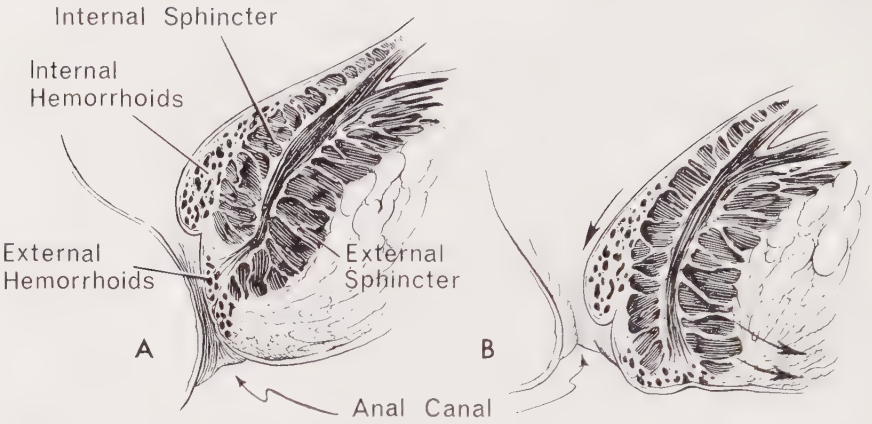

Figure 4. *A*, Relative position of sphincter muscles and hemorrhoids, without anesthesia. *B*, Changes in position of sphincter muscles and hemorrhoids with traction or at the time of defecation.

expand, contract and change in shape. Thus, the rectum and anal canal are not fixed in position. Figure 4, *A*, is representative of the work of Eisenhammer,[2] Goligher[3] and Brossy[1] on the sphincter muscles at rest and the relative position of the internal and external hemorrhoids. Figure 4, *B*, demonstrates the change of position of the sphincters in a patient under anesthesia, during surgery, especially with traction, and probably at the time of defecation.[3] Note in Figure 4, *B*, how the external sphincter moves laterally away from the canal and the internal sphincter moves down and out to a more superficial position. It is the internal sphincter that is incised when a fissurectomy or a hemorrhoidectomy is completed and not the subcutaneous portion of the external sphincter. Traction, under anesthesia, further displaces downward the position of the internal sphincter. Following recovery from surgery, the sutured hemorrhoidal stump or fissure bed retracts upward.

SUMMARY

There is still confusion in the clinical differentiation and management of hemorrhoids, anal fissures, perianal abscesses and fistulas. In this article I have attempted to clarify these common ailments and outline some of the basic principles involved.

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An Anatomic Reappraisal of the Posterior Inguinal Wall

Special Consideration of the Iliopubic Tract and Its Relation to Groin Hernias

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The anatomy of the lower abdominal wall, particularly where the abdominal musculature and root of the thigh join, has been described interminably. Many of these works are excellent (Anson, Morgan and McVay, 1960; Clark and Hashimoto, 1946; Condon, 1964; Fruchaud, 1956; Griffith, 1959; Lytle, 1945; and McVay and Anson, 1940) and should be reviewed by all interested in this anatomic area. It would seem redundant to review this subject again, except that there are certain aspects of this anatomy which have not been stressed sufficiently. This point of view developed following recent groin dissections during autopsy surgery. Our concepts have been solidified during the performance of over 800 hernial repairs wherein the posterior inguinal wall was visualized, dissected, and repaired by the preperitoneal approach (Nyhus, 1964a).

This paper will not attempt to redescribe groin anatomy in its entirety; rather, those structures which seem to have a position of prime importance in the etiology and repair of hernial defects have been selected for further discussion.

ANATOMY

It is our concept that the posterior lamina of the inguinal abdominal wall (transversus abdominis–transversalis fascia stratum or lamina) (Fig. 1) is the all-important layer in both the etiology and the repair of groin hernias. The inguinal ligament is a part of the anterior lamina (external

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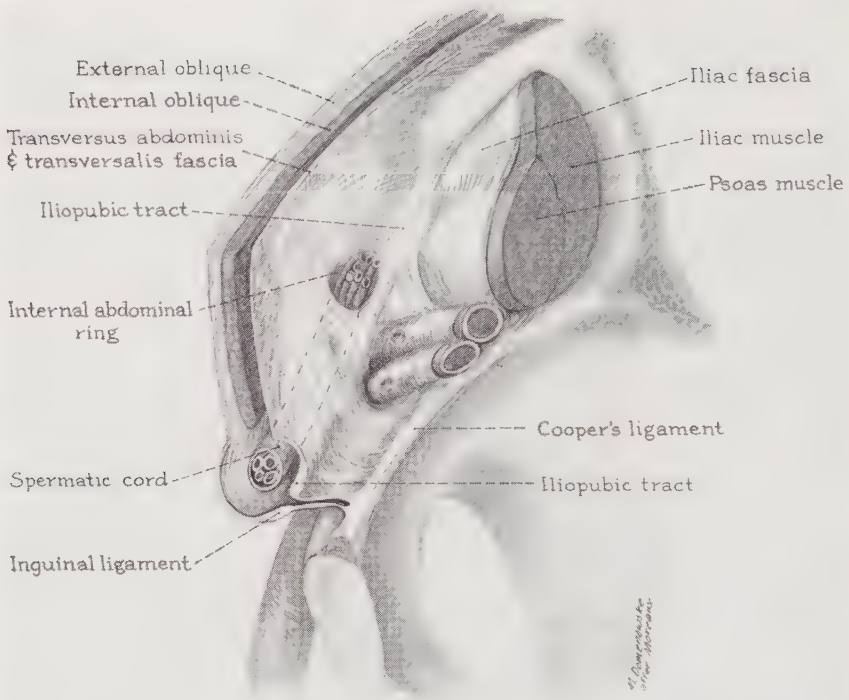

Figure 1. Fasciomusculoaponeurotic components of the lower abdominal wall. Right posterior oblique view. Note the relationship of the transversalis fascia to the structures in the posterior inguinal wall. (Modified from Fruchaud, 1956.) (*Note especially:* All six figures in this paper show the *right* side.)

oblique aponeurosis–innominate fascia stratum or lamina) and as such is of no consequence to either the etiologic mechanisms of hernial development or the modalities of hernial repair.

Transversalis Fascia

This is a part of the endoabdominal fascia that lines the entire peritoneal cavity; it takes its name from the adjacent transversus abdominis muscle and aponeurosis. This internal investing fascia of the transversus abdominis separates the muscular abdominal wall from the preperitoneal fat. As a portion of the endoabdominal fascia, the transversalis fascia is immediately continuous with the lumbar, iliac, psoas and obturator fasciae. Medially, it continues as the rectus fascia covering the posterior part of the rectus muscle. Finally, it meets the transversalis fascia from the opposite side.

The transversalis fascia is not a solid sheet of tough fascia which is readily available in all areas for hernial repair. Rather, it waxes and wanes in its quantity and quality. Fortunately it waxes or accompanies

aponeurotic structures in key areas. Thus, it has gained a reputation for availability which probably is warranted. These special parts of the transversalis fascia and its accompanying aponeurotic structures include: (1) iliopubic tract; (2) transversalis fascia crura and sling of the internal inguinal ring; and (3) arch of transversus abdominis aponeurosis.

Iliopubic Tract (*Ligamentum Iliopubicum Thomsonii, Bandelette Iliopubienne, Deep Crural Arch, Deep Femoral Arch, Anterior Femoral Sheath*) (Figs. 1, 2, 3, 6). This is a strong fascial band which begins laterally along the crest of the ilium and at the anterior superior iliac spine. In this area it gives origin to the iliacus muscle and the lowermost fibers of the transversus abdominis muscle. The iliopubic tract arches over the psoas muscle and the femoral vessels, where it forms an integral part of the anterior femoral sheath. In its midportion it lies immediately subjacent to the inguinal ligament. It is, however, completely separated from the

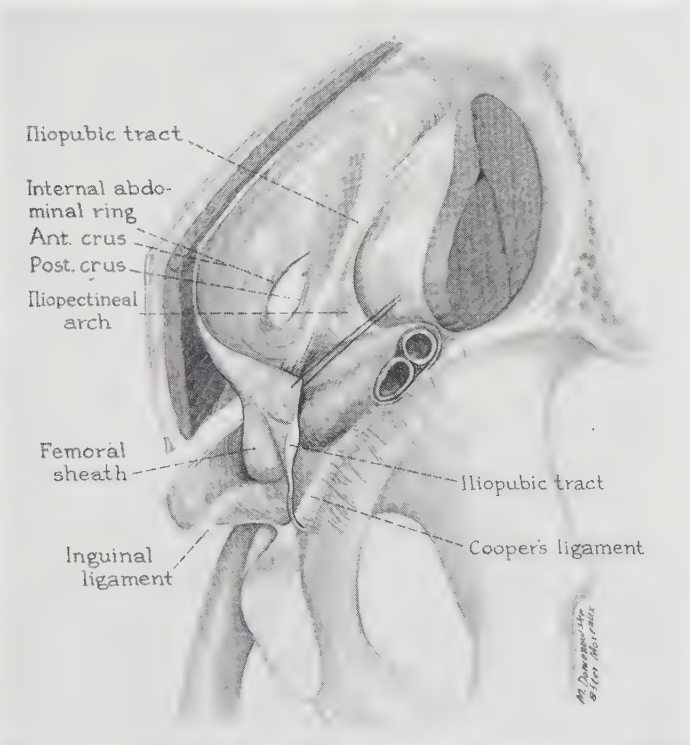

Figure 2. Fasciomusculoaponeurotic components of the lower abdominal wall (*continued*). Similar view to Figure 1. Spermatic cord has been removed and the iliopubic tract is reflected to demonstrate fascial continuity with the femoral sheath. The posterior inguinal wall (transversus abdominis–transversalis fascia lamina) is firmly "rooted" to the thigh at this point. The crura of the internal abdominal ring also are well shown. (Modified from Fruchaud, 1956.)

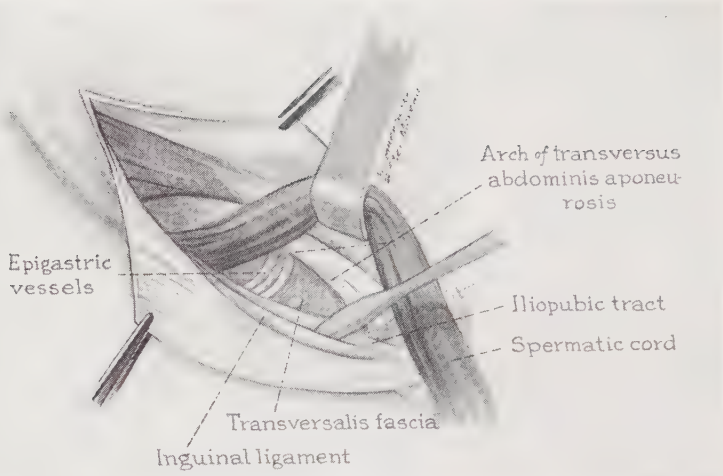

Figure 3. Anterior view of inguinal canal. The external oblique aponeurosis has been split and reflected. Note relation of iliopubic tract to the inguinal ligament. (Modified from Fruchaud, 1956.)

inguinal ligament and the relationship is one of proximity only.* Continuing medially, the iliopubic tract inserts fanwise into the superior ramus of the pubis and into Cooper's ligament. The most inferior fibers of the iliopubic tract—those which insert most laterally into Cooper's ligament are sharply recurved in the normal groin. It is this recurved portion of the iliopubic tract which defines the medial border of the femoral canal, not the lacunar (Gimbernat's) ligament of classic description.

The fundamental importance of the iliopubic tract in understanding the anatomy of groin hernias is appreciated when one realizes that the iliopubic tract forms one of the margins of the hernial defect in each of the common groin hernias. Direct and indirect inguinal hernial defects are limited on their posterior aspects by the fibers of the iliopubic tract; femoral hernial defects are similarly limited on their medial and anterior aspects.

Transversalis Fascia Crura and Sling of the Internal (Inguinal) Ring (Fig. 2). The internal ring is given an elliptic shape by the presence on

* This anatomic structure was first described by Alexander Thomson (frequently misspelled Thompson) in 1836. Thomson recognized this important thickening in the posterior inguinal wall as an entity distinctly separate from the inguinal ligament. He wrote: "There is not one single fiber coming from the external oblique muscle. . . . We always find behind the tendinous portion of the external oblique, which forms Poupart's ligament, a strong aponeurotic bandelette, which has been, but wrongly, confused with the tendon itself."

Eugen Polya (1912)—of Polya gastrectomy fame—also was a strong proponent of the individuality of the iliopubic tract. Polya made several interesting comments concerning this structure, among them the following: "The opinions are not unanimous whether the ligament is part of the transversalis fascia, Poupart's ligament, femoral sheath or the psoas fascia. As far as I am concerned, and most of the other authors agree, this ligament constitutes a part of the transversalis fascia."

its anterior and posterior aspects of a double fold of transversalis fascia which cradles and supports the spermatic cord in this region. The anterior crus is the longer, being attached to the transversus abdominis above the internal ring by the interdigitating fascial slips which lace together the investing fasciae on the two surfaces of that muscle. The posterior crus or arm of this sling consists of fibers which parallel the iliopubic tract and finally fuse with it. The two crura are, of course, continuous with each other on the medial aspect of the internal ring and the spermatic cord, and, since they are more prominent at this level, a sling effect is given; thus the terminology, transversalis fascia sling of the internal inguinal ring.

This fascial sling is considered to be of importance as a shutter mechanism which partially closes the internal inguinal ring during periods of increased intra-abdominal pressure (Lytle, 1945). The fascial sling and crura are of importance to the surgeon in the repair of indirect hernias. Close approximation of the transversalis fascia sling around the cord structures at the internal inguinal ring is the essential maneuver in this repair. The sling delineates the margins of the defect and facilitates accurate placement of the sutures between the transversalis fascia transversus abdominis aponeurosis components of this anterior crus and the transversalis fascia–iliopubic tract components of the posterior crus.

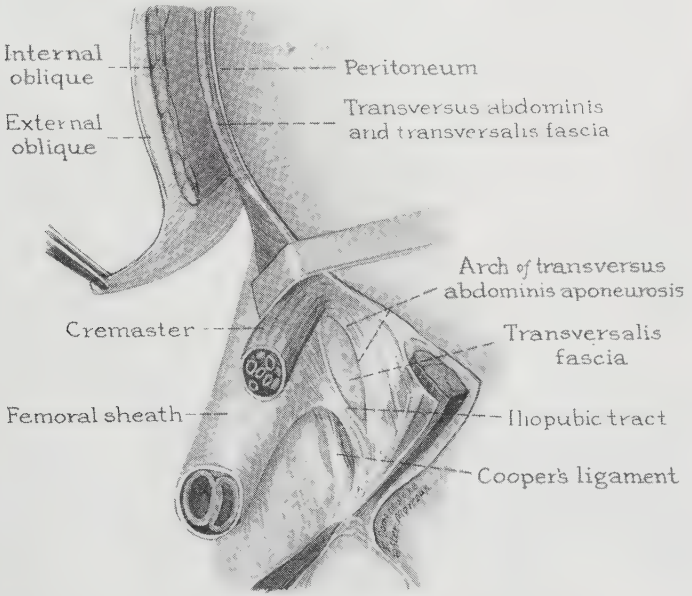

Figure 4. Right anterior oblique view of lower abdominal wall. The external oblique and internal oblique muscles and aponeuroses have been cut and/or reflected. The relation of the transversus abdominis–transversalis fascia stratum to other structures of that layer is seen anteriorly. (Modified from Fruchaud, 1956.)

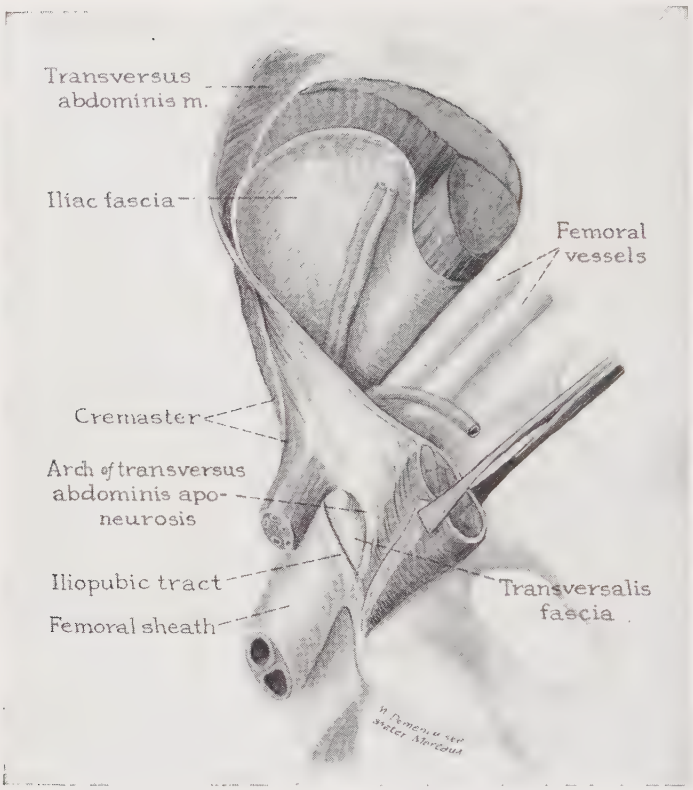

Figure 5. Continuity of transversalis fascia and transversus abdominis aponeurosis with iliac fascia, femoral sheath, and iliopubic tract. Inside-outside view of right posterior inguinal wall. The cremaster muscle and fascia originate from the internal oblique muscle and interparietal fasciae. (Modified from Fruchaud, 1956.)

Arch of Transversus Abdominis Aponeurosis (Figs. 3, 4, 5). The aponeurosis of the transversus abdominis muscle forms a well-defined curved arch which serves as an important landmark for the surgeon. This structure serves as the superior limiting border of most direct hernias. As indicated previously, the transversalis fascia is intimately attached to this aponeurosis and therefore is not readily separated from it. This observation has clinical importance. The superior edge of a direct hernial defect will consist of both transversalis fascia and aponeurosis of transversus abdominis. The main strength of the suture placed at the superior edge of the direct defect will result from use of this aponeurotic arch of transversus abdominis.

CONJOINED TENDON. The fusion of the aponeuroses of the internal oblique and transversus abdominis muscles to form one structure is the exception rather than the rule. The phenomenon occurs in less than 10 per cent of dissected specimens. Thus, the term conjoined tendon should be discarded in favor of more specific terminology.

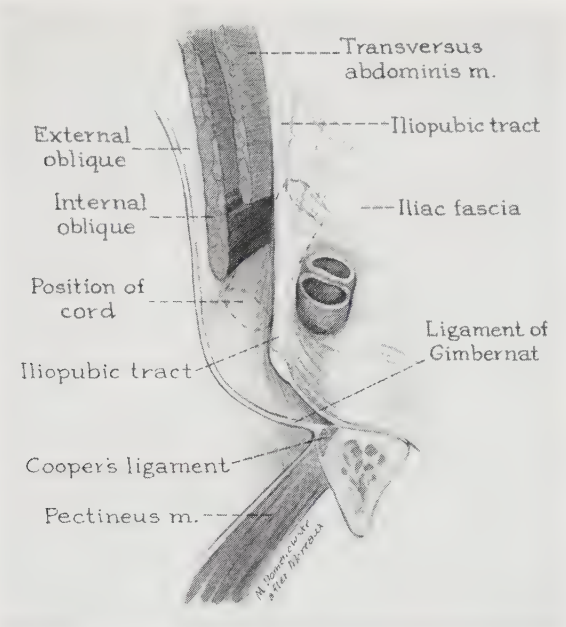

Figure 6. Relation of the iliopubic tract to the femoral vessels and to Gimbernat's ligament. Medial cross section of right lower abdominal wall, viewed medial to lateral. (Modified from Fruchaud, 1956.)

OPERATIVE OBSERVATIONS

Approach

The repair of the posterior lamina of the inguinal wall which utilizes the iliopubic tract as a major structure has many advocates. The majority of authors have preferred the anterior approach (Clark and Hashimoto, 1946; Donald, 1948; Griffith, 1959). Nyhus (1964a, 1964b) has advocated the preperitoneal approach for the iliopubic tract repair. The posterior lamina and the important thickenings of transversalis fascia (iliopubic tract and transversalis fascia sling of the internal inguinal ring) are seen directly by the latter approach. These same structures are seen clearly only after excision of the cremaster muscle, when an indirect inguinal hernia is approached anteriorly (Griffith, 1959). This maneuver is not well recognized and many so-called *plastic repairs* of the internal abdominal ring must be less than satisfactory because of this failure to delineate the proper fascial margins of the ring. Visualization of the posterior abdominal wall lamina during an anterior repair of a direct hernia is relatively simple, assuming the operator is cognizant of, and looks for, the iliopubic tract.

The Problem of Nomenclature

One of today's most respected scholars in the field of groin anatomy and surgery is Chester B. McVay of Yankton, South Dakota. Certainly

his writings have stressed the importance of transversus abdominis stratum or lamina in the repair of groin hernias. Yet, in none of his presentations is the iliopubic tract mentioned. Careful scrutiny of some of the drawings in his monograph (McVay, 1954) suggested to this author that the structure labeled "femoral sheath" was identical to the structure we call "iliopubic tract." Recently we had the pleasure of reviewing a film on inguinal hernioplasty (McVay, 1959) which clarified the problem immeasurably. The anterior femoral sheath was shown in a very clear manner, and it also was used as a portion of the repair depicted. It is the firm conviction of this observer that the anterior femoral sheath, as shown in this film, is the same structure that we call the iliopubic tract. Therefore, the problem seems to be principally one of semantics.

It appears that we are in basic agreement with McVay concerning all the structures of the transversus abdominis stratum, including the femoral sheath or iliopubic tract. The use of these structures by the Yankton group, in the repair of various types of groin hernias, varies somewhat from the Seattle group's methods. It is our belief that the iliopubic tract may be used in conjunction with arch of transversus abdominis aponeurosis (direct hernia), anterior crus of internal abdominal ring (indirect hernia), and Cooper's ligament (femoral hernia) during the various hernial repairs. Cooper's ligament plays a more prominent role in the Lotheissen-McVay repair. The Cooper's ligament repair has proved to be excellent when used according to proper indications. The preperitoneal approach and the iliopubic tract method of repair continue to be evaluated both as to indications for use of the approach and long-term results of the repair. Since the same basic tissues (transversus abdominis lamina) are used in both hernial repairs, the final comparison of results between the two methods will be of exceptional interest.

SUMMARY

The importance of the posterior inguinal wall to the etiology and repair of groin hernias is stressed. The most important components of this transversus abdominis–transversalis fascia layer are: (1) iliopubic tract; (2) transversalis fascia crura and sling of the internal inguinal ring, and (3) arch of transversus abdominis aponeurosis. The "conjoined tendon" is infrequently present and is of little practical value.

The iliopubic tract method (posterior inguinal wall) of hernial repair may be used with either an anterior or posterior operative approach. Each approach has its advantages and disadvantages. The anterior Lotheissen-McVay approach has proved to be of great value. The preperitoneal approach and iliopubic tract method of repair continue to be evaluated.

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The Surgical Anatomy of the Adrenal Glands with Particular Reference to the Suprarenal Vein

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The main surgical procedure carried out upon the adrenal glands is still their ablation, either of one adrenal alone for tumors involving the cortex or medulla, or both in order to modify hormonal effects. Elusively placed in the upper and posterior part of the abdominal cavity, it is not surprising that until 1563 when Eustachius described them, their existence was not known. Until a satisfactory substitute for the normal hormone was available, removal of the adrenal was limited to those containing tumors, in which the normal anatomy is usually distorted. With cortisone available, the removal of anatomically normal glands is commonplace, and this has stimulated reinterest in their anatomy. Most of this has related to methods of approaching the glands, their situation and sites where accessory adrenal tissue may be found, and their blood supply, particularly the venous drainage, the securing of which is the principal step in the removal of the gland.

NORMAL SITUATION

Both glands lie in the fat in relation to the upper and medial aspects of the superior pole of the kidney on either side (Fig. 1). The left is a bit bigger than the right, the usual size being 3 to 5 cm. in height and 2 to 3 cm. in breadth. The normal weight is 7 to 12 grams. In patients who have been on adrenal corticoids for some time, the size may be deceptively small, and the gland more difficult to identify. In color they are usually yellow, but it may be difficult to see them in the fat. On the left side, the

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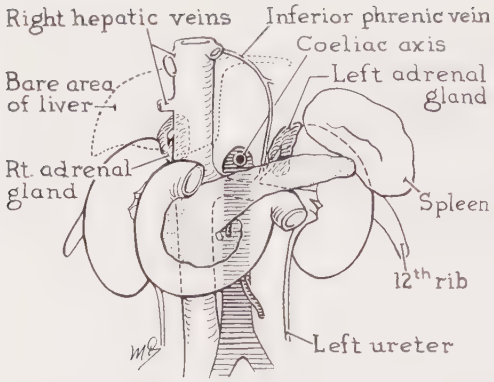

Figure 1. Normal situation and relations of right and left adrenal glands.

vein draining into the renal vein is usually evident, and will lead the operator to the gland, but on the right side this is not so, and it is often better to palpate the firmer gland in the fat, and clear one edge when the characteristic yellow, sharp, rounded, finely lobulated structure can be seen and thus identified.

The right gland is perhaps more triangular. It lies within the renal fascia mostly behind the inferior vena cava on the right crus of the diaphragm as this attaches to the upper lumbar vertebrae. The upper part of the portion lateral to the vena cava lies behind the posterior surface of the liver, the "bare area," where the liver is in direct contact with the diaphragm. Below and laterally it is covered by peritoneum of the posterior abdominal wall which passes from the duodenum to the front of the kidney, over the suprarenal, to the lower surface of the liver. As a result, the duodenum may be in anterior relation to the lower anterior surface of the right suprarenal, and above, the lowest right hepatic vein may be close as it passes to the vena cava covered by the hepatorenal reflection of the peritoneum. The lateral and inferior edges of the right suprarenal are generally visible, but the upper medial edge and the upper pole are hidden behind the vena cava. The hilus of the gland is on the anterior surface about its middle, and from here emerges the short adrenal vein.

The left gland lies along the medial border of the left kidney at its upper pole. It is more elongated, and its hilus is on the anterior surface, the vein lying in rather a groove and emerging at the lower end. It is covered by the peritoneum of the posterior wall of the lesser sac, and lies on the left crus of the diaphragm, a little distance away from the aorta. The tail of the pancreas, and the splenic vessels may be related to the lower part of its anterior surface.

SURGICAL APPROACH

The surgical approach to the adrenal glands is a choice between adequate exposure for one gland or a less adequate simultaneous exposure

for both. Lying as they do in the upper and posterior recesses of the abdomen, they are protected by the rib cage from all except an anterior transabdominal approach, unless ribs are resected. If an anterior approach is chosen, the distance to be traversed before the adrenals are reached may be great, especially in obese patients, and if the adrenal is enlarged, space to maneuver may be limited. However, such an approach does afford the opportunity to explore the abdominal cavity, carry out additional procedures, such as oophorectomy, and examine both adrenals and sites where accessory tissue may exist, through the same incision.

The most convenient anterior exposures are obtained through either a transverse incision above the umbilicus, or an oblique incision, 10 cm. below the right costal margin and parallel to it, extending from the left costal margin to the right flank, or a bilateral subcostal inverted "V" (Fig. 2). In all, exposure is normally adequate and the wounds heal well.

After the abdominal cavity has been opened, the left adrenal may be approached in several ways. The lesser sac may be opened by dividing the gastrocolic omentum along the greater curve of the stomach, distal to the gastroepiploic vessels. The pancreas can then be displaced downward, and the peritoneum and renal fascia opened where they lie over the adrenal (Fig. 3, *A*). The left adrenal can also be approached through the bloodless area of the mesentery of the transverse colon, at the root of its left half, between the middle colic and left colic vessels. This incision opens the retroperitoneal space over the pancreas, which has to be elevated to expose the adrenal (Fig. 3, *B*). The third alternative is to mobilize the spleen and tail of the pancreas on their lateral side, and reflect them medially. This is much easier to do in the cadaver than at operation, for in practice bleeding from the bed can be very troublesome and no better exposure is obtained (Fig. 3, *C*).

On the right side, the gland is harder to find. The hepatic flexure of the colon must be displaced downward, the right kidney identified by palpation, and the peritoneum of the posterior abdominal wall at its upper pole cleared. When this is done the vena cava will be obvious medially, with the liver lying above and the duodenum below and medial. The liver should be retracted upward, and keeping the kidney drawn downward the

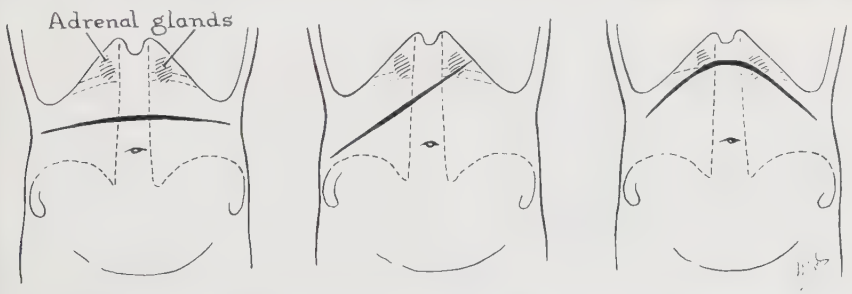

Figure 2. Abdominal incisions suitable for anterior abdominal approach.

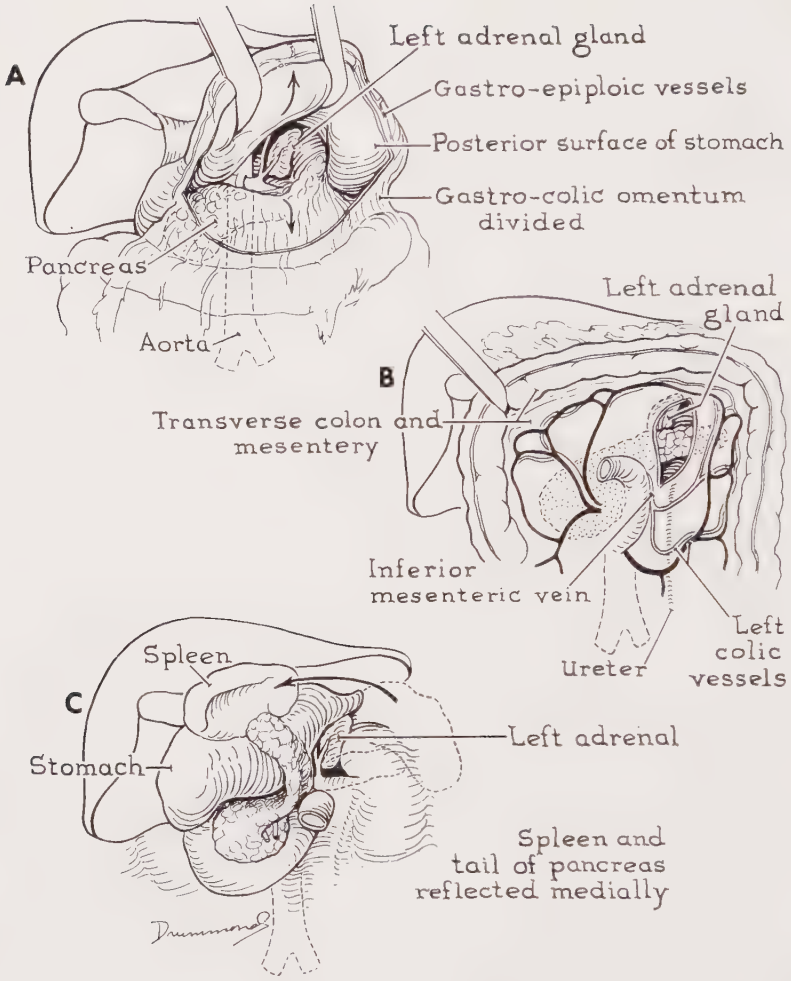

Figure 3. Intra-abdominal exposure of left adrenal gland: *A*, through gastro-colic omentum; *B*, through transverse mesocolon; *C*, by mobilization of spleen and tail of pancreas.

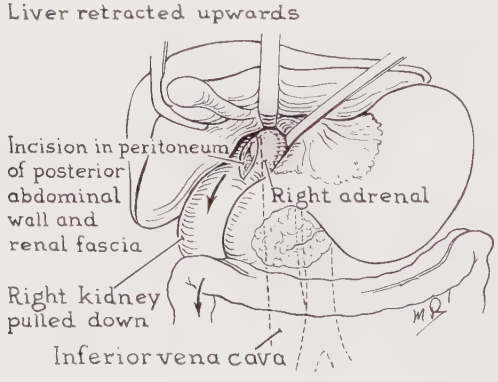
Liver retracted upwards
Incision in peritoneum of posterior abdominal wall and renal fascia
Right adrenal
Right kidney pulled down
Inferior vena cava

Figure 4. Intra-abdominal exposure of right adrenal gland.

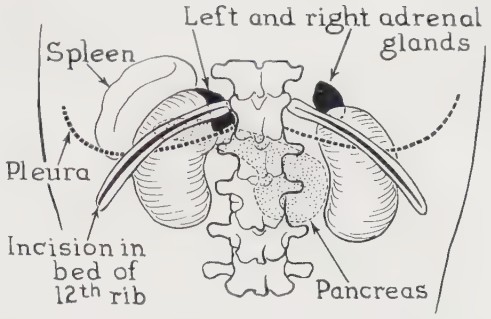

Figure 5. Posterior approach to adrenals through the bed of the twelfth rib.

peritoneum and renal fascia cranial to the upper pole incised (Fig. 4). The perirenal fat is seen, and usually the lateral and inferior edge of the adrenal can be identified, the remainder lying behind the vena cava and palpable against the vertebrae. It is generally higher and more posterior than one expects.

If the adrenal is to be approached posteriorly, it is usual to do this through the bed of the twelfth rib, after the rib has been resected (Fig. 5). The pleura is in close relation to the medial half of the twelfth rib, and may be injured if it is not reflected upward. The kidney can be palpated through the posterior layer of renal fascia. It is displaced downward, the fascia divided above the upper pole, and the adrenal found in the perirenal fat, where it can be rotated medially to display the vein after the lateral and upper and lower borders have been cleared. On the left side the close proximity of both spleen and the tail of pancreas should be remembered.

Such a posterior approach is usually inadequate for large tumors, and better exposure is obtained by a combined thoracoabdominal incision through the ninth interspace, the incision being continued onto the anterior abdominal wall dividing the costal cartilage, and the rectus sheath and muscle to the midline if necessary. The pleural space is opened and the diaphragm is divided laterally from before backward to a short distance from the spine. If the incision is kept lateral the inferior phrenic vessels can be avoided and the diaphragm retains its innervation intact. The peritoneum underlying is not opened, but is displaced forward. The retroperitoneal space containing the upper pole of the kidney, the adrenal and vena cava (or aorta) can then be displayed.[7]

ABNORMALITIES OF POSITION

One adrenal may be absent in which case the other will be unusually large, or they may be of different size. Fusion of both the adrenals across the midline, behind the vena cava and aorta, has been described. Of more importance is the occurrence of accessory adrenal tissue which may be ectopic. An understanding of the sites where accessory tissue may be found is more easily grasped by a knowledge of their embryology.[2, 4, 8]

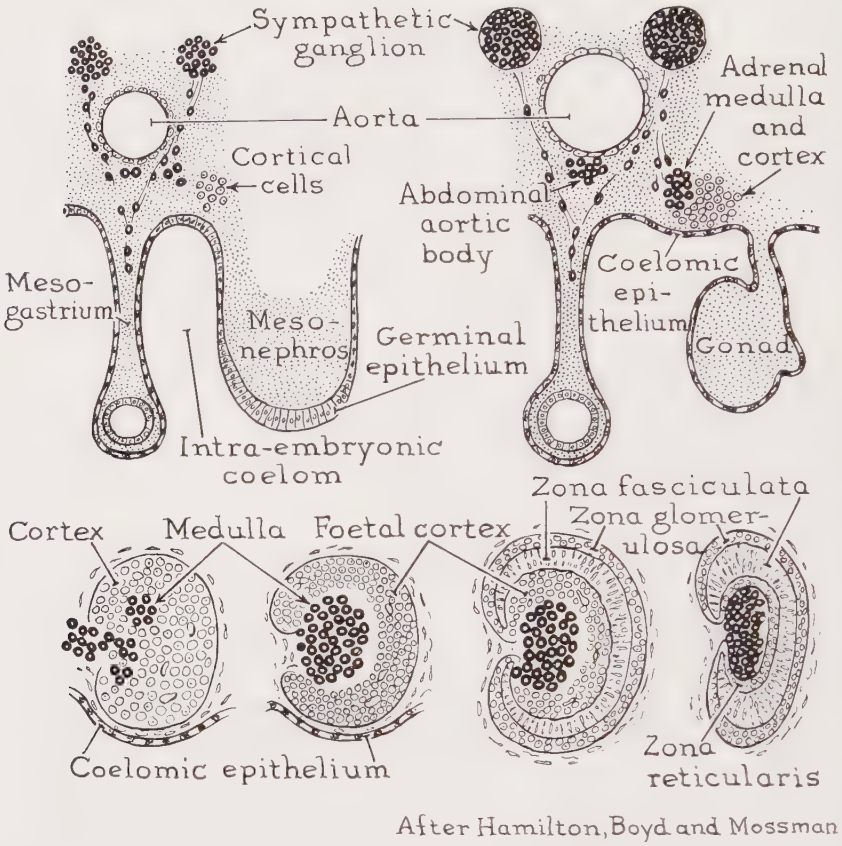

Figure 6. Development of cortex and medulla of adrenal.

The cortex and medulla arise from different embryological sources. In some lower vertebrates these may remain separate. In man the two elements are fused in the adrenal gland, but accessory tissue may consist of separate cortex or medulla, or may consist of both elements.

The cortex develops from the columnar mesothelial cells of the intra-embryonic coelom situated in the adrenal groove between the root of the mesogastrium medially and the urogenital ridge laterally at the level of the mesonephros (Fig. 6). The cortical cells, as they differentiate from the coelomic mesothelial cells, become embedded in the neighboring mesenchymal tissue, and a capsule forms about them (7 mm., 20 days). These cells give rise to the fetal cortex. Later, further cells develop from the adrenal groove which are smaller in size and overgrow the fetal cortex and penetrate the capsule to intermingle with the fetal cells (12 mm., 42 days). These cells will form the permanent cortex. The relatively bulky fetal cortical cells have usually disappeared by the first year of life, and only the permanent cortical cells persist.

The medulla develops from the sympathetic neuroblasts originally

derived from the neural crest, which first form in the thoracic region and extend into the cervical and lumbosacral regions. These sympathetic cells, which appear on the medial dorsal aspect of the primitive cortex, invade it (14 mm., 44 days) and reach the central vein (19 mm., 49 days). Eventually they are completely encapsulated by the cortex at a much later date of intraembryonic life. The medulla is part of the widely distributed chromaffin system and it is better to consider accessory medullary tissue as part of this system, rather than the remainder of the system as accessory adrenal medulla. Chromaffin tissue includes the paraganglia associated with ganglia of the sympathetic trunk and the sympathetic plexuses, particularly about the coeliac axis; the aortic bodies, lying in front of the lower aorta near the origin of the inferior mesenteric artery; and other chromaffin tissue in relation to the viscera, including kidney, ureter, prostate, epididymis, ovary and paraoopheron.

The sites of accessory cortical tissue are related to the structures which develop from tissue in the vicinity of the adrenal cortical groove, particularly the mesogastrium and the urogenital ridge, and which may carry with them rests of adrenal cortical tissue. Most frequently this accessory tissue is found in the vicinity of the main glands, about the coeliac axis, and represents cells sequestered in the mesenchymal tissue at the root of the dorsal mesogastrium. Graham[3] showed that, of 100 consecutive autopsies, 32 showed accessory tissue in this area averaging $7 \times 4 \times 3$ mm.; and of these, 16 were comprised of both medullary and cortical tissue. Cortical tissue here has also been found under the capsule of the right lobe of the liver.

With the migration of renal and gonadal tissue developed from the urogenital ridge, accessory cortical tissue has been found embedded in the upper pole of the kidney and related to the lower pole, along the course of the gonadal vessels, in or near the spermatic cord, in the mediastinum testis and tunica testis, in the connective tissue between testis and epididymis, around the tail of the epididymis, within the epididymis and in the broad ligaments and the ovary. In most cases the tissue has been microscopical, and is more frequently found in the newborn and infants. The actual incidence of accessory cortical tissue in adults is hard to determine, but is probably frequent, though the size of the tissue is usually very small.[6] It does not seem that these accessory nodules become large after adrenalectomy, when replacement hormonal therapy follows. Cortical tumors have been found at these sites.

THE BLOOD VESSELS OF THE SUPRARENAL

With increasing familiarity with adrenalectomy it has become apparent that the textbook description of the blood vessels of the adrenal gland is inadequate. As the greatest hazard to the operation of adrenalectomy is

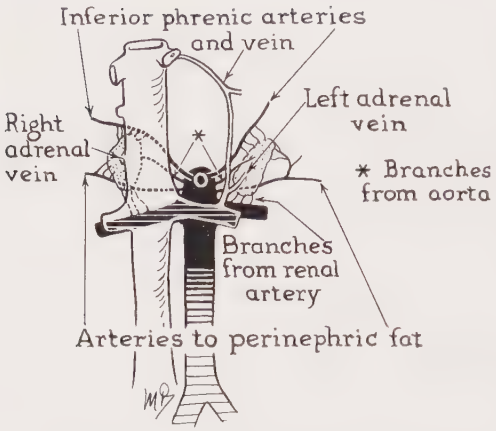

Figure 7. Arterial supply of adrenals.

bleeding, a more detailed knowledge of these vessels is required. In particular, this is so for the adrenal veins. Major bleeding is mainly venous, either from the adrenal veins or the large veins into which they drain, which may be inadvertently torn by avulsion of an unrecognized anomalous vein. Arterial haemorrhage is seldom a problem. The three large arteries of textbook description which are said to supply the gland are seldom found. A review of current anatomical description is important.[1]

Arterial Supply

On both sides the arterial blood comes from the inferior phrenic artery, the renal artery and the aorta (Fig. 7). However, seldom are there single vessels from each of these sources, but rather the glands lie at the axis of a cartwheel, the spokes being the numerous small arteries coming from the periphery as branches of these main vessels. By the time they reach the borders of the adrenal they have broken up into many small arterioles, or there may be up to eight or nine small branches from the parent artery passing in parallel strands to the gland. The inferior phrenic artery usually supplies the major part of the blood. Because of their small size the arterioles seldom cause severe bleeding when divided close to the gland, though often a moderate sized branch from the aorta to the left gland may cause trouble.

Venous Drainage

The veins draining the gland are much more constant.[5] In injection dissection preparations specifically designed to display the suprarenal veins in ten subjects, the right vein was single in eight, and joined the inferior vena cava in five (Fig. 8). In these, the trunks, on the average, were under 0.4 cm. in diameter, and joined the vena cava 3.6 cm. cranial to the right renal vein, the trunk being short (on the average 1 cm.). In the other three, the single vein joined a right hepatic vein close to the junction of

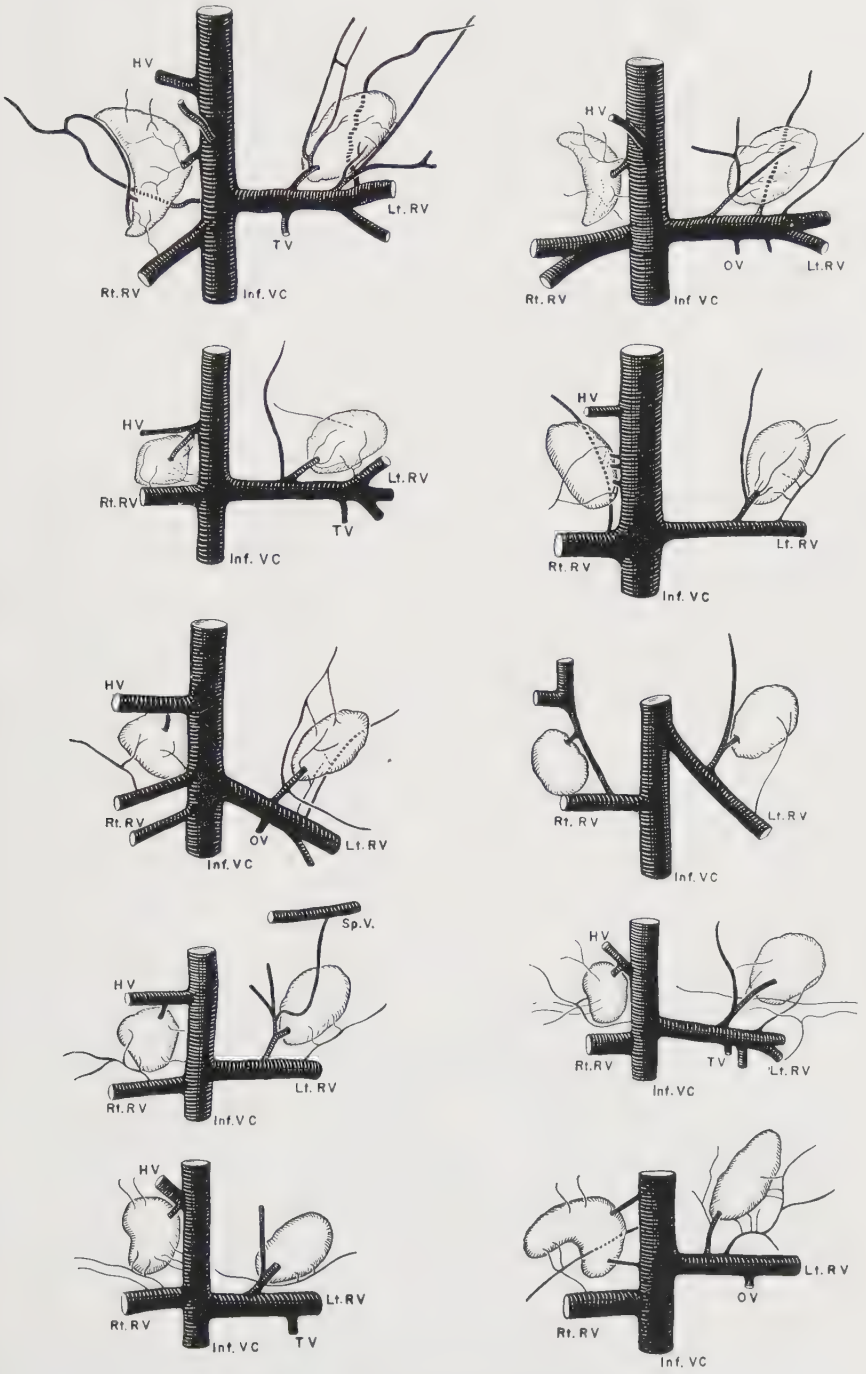

Figure 8. Venous drainage of adrenals

the latter with the inferior vena cava. In these subjects, the right hepatic vein was within 4.0 cm. of the right renal vein. In one subject there were two right veins, and in another three, all of which joined the inferior vena cava between the lowest hepatic vein and the right renal vein. In addition to these main veins, small venules from the capsule passed to the many other neighboring veins, viz. main suprarenal, perirenal, renal, inferior phrenic, peritoneal and the inferior vena cava. The left suprarenal vein was single in all subjects and after receiving the left inferior phrenic vein had an average diameter of over 0.4 cm. It invariably joined the left renal vein at an average distance of 3.1 cm. from the junction of the latter over the inferior vena cava, and opposite or medial to the gonadal vein. The junction with the left inferior phrenic vein took place either close to the renal vein or within an average of 1.6 cm. distal to the renal vein. In one subject the left inferior phrenic vein did not join the suprarenal vein. In addition, small capsular veins pass to the main suprarenal vein, perirenal fat veins, inferior phrenic vein, renal vein, and ascending lumbar vein.

The practical importance of this is the necessity to display the superior, medial and inferior borders of the right gland carefully in order to discover the relatively common anomalous pattern of drainage. A tear may involve the side of the vena cava or an hepatic vein. If this occurs the patient can be placed head down, the flow in the vena cava temporarily controlled by pressure caudal to the tear, and the portion of the vena cava torn isolated with a Satinsky clamp, and sutured.

On the left side, if the adrenal vein is ligated close to the left renal vein, it should be remembered that the inferior phrenic vein joins the adrenal vein proximal to the ligature and will require to be ligated as well if bleeding is to be avoided.

SUMMARY

The situation and relationships of the adrenal glands are described, and indications and techniques of approach given for the transabdominal, posterior and thoracoabdominal routes. Anomalies of position and sites of ectopic cortical and medullary tissue are discussed with reference to their embryological development.

The normal blood vascular supply is described with an account of venous anomalies which may be encountered.

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Surgical Anatomy of the Urinary Bladder and Pelvic Ureter

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URINARY BLADDER

The increasing frequency and scope of pelvic operations make knowledge of the urinary bladder and its relationships with other viscera important alike to urologist, gynecologist and general surgeon.

Relationships

With the patient in the supine position the empty bladder lies at about the level of the symphysis pubis. As the bladder distends it rises cephalad out of the pelvis to become, for practical purposes, an intra-abdominal organ, even though it remains extraperitoneal.

In the female the bladder rests directly upon the pelvic diaphragm. In the male the prostate gland is interposed between the bladder neck and the pelvic diaphragm. Anteriorly, the bladder is separated from the symphysis pubis by the prevesical space, occupied by areolar tissue. Superiorly, the viscus is covered by peritoneum, freely movable except for an adherent patch near the posterior portion of the dome. Laterally, the boundaries are the muscles and fascia lining the pelvis. Posteriorly, in the male, the seminal vesicles and the vasa deferentia with their ampullae lie beneath the floor. In the female, the ureterine corpus indents the posterosuperior aspect of the bladder. Below this, the vagina is closely related to the floor.

The base of the bladder is fixed by its attachment to the prostate in the male and the urethra in the female. Anteriorly, these structures are fixed to the undersurface of the pubis by the puboprostatic, pubourethral and pubovesical ligaments. These condensations of the pelvic fascia are particularly strong in the male where they form two heavy bands (medial puboprostatic ligaments) fastening the prostate to the pubis. Similar condensations of pelvic fascia form the lateral puboprostatic ligaments which

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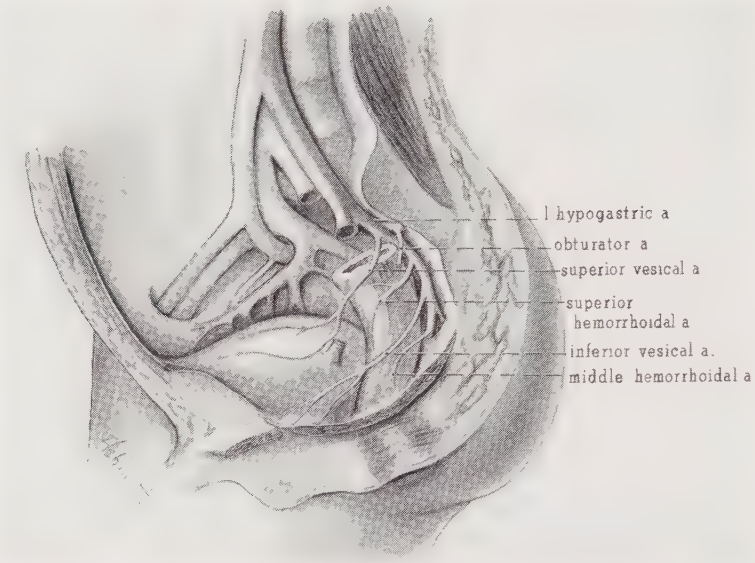

Figure 1. Arterial supply of the left side of the urinary bladder.

run between the fascia covering the levator ani muscles and the fascia surrounding the prostate and bladder neck. Posteriorly, the floor of the bladder and prostate in the male are fused to the anterior wall of the rectum by Denonvillier's fascia. Anteriorly, the bladder is attached to the anterior abdominal wall by the median umbilical ligament (urachus), running from the vertex of the bladder to the umbilicus on the posterior aspect of the anterior abdominal wall, and the lateral umbilical ligaments which are the obliterated extensions of the superior vesical artery. These maintain the application of the bladder to the anterior abdominal wall as it distends and rises out of the pelvis.

A double fold of peritoneum is draped over the anterior surface of the bladder. This rides up as the bladder distends or it may be wiped upward manually at surgery, exposing the anterior aspect of the bladder which may be approached extraperitoneally.

Blood Supply and Drainage. The blood supply of the bladder (Fig. 1) is derived from two branches of the anterior division of the hypogastric artery. The superior vesical artery supplies the anterior and lateral portions. The inferior vesical artery supplies the fundus and base and sends branches to the terminal portion of the ureter, the seminal vesicles and the prostate gland. In the female, this artery supplies branches to the vagina.

The venous drainage of the bladder (Fig. 2) is made up of the plexus of Santorini, anterior to the prostate in the male and the bladder neck in the female, and the lateral vesical plexus on either side of the bladder neck. A veritable network of small veins runs laterally to coalesce into the hypogastric veins.

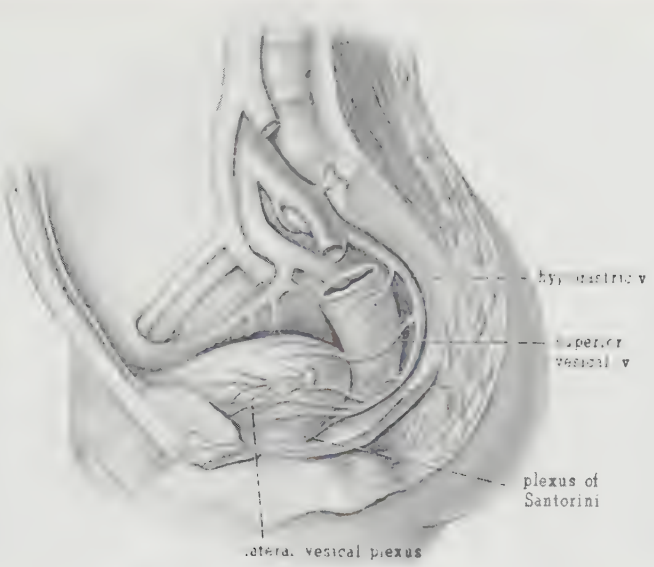

Figure 2. Venous drainage of the urinary bladder, left side.

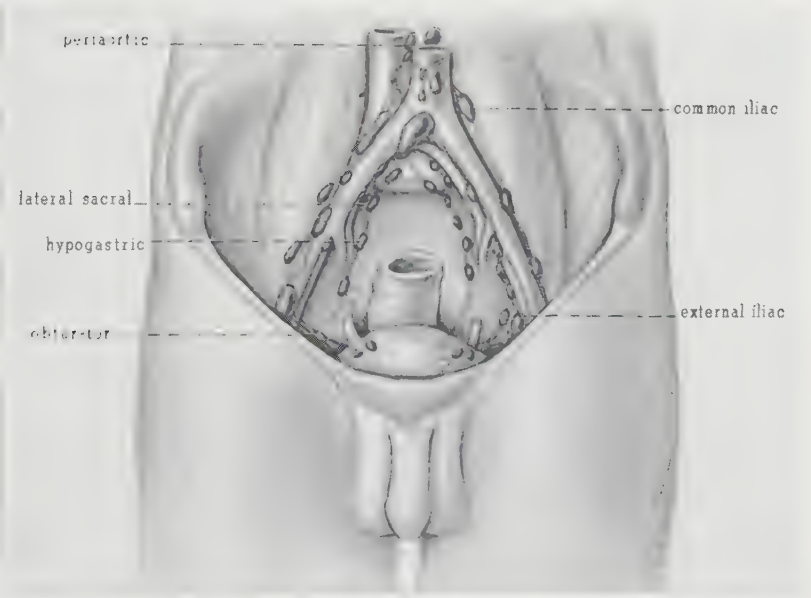

Figure 3. Lymphatic drainage system of the urinary bladder.

Lymphatics. Lymphatic channels (Fig. 3) from the trigone and base course laterally to the obturator fossa nodes and to nodes along the external iliac vessels. Channels from the dome and lateral walls drain into nodes

along the hypogastric vessels. They follow these vessels upward to drain into the common iliac and periaortic nodes.

Innervation. The important motor and sensory innervation of the bladder arrives via the pelvic nerve and plexus from the parasympathetic outflow from sacral nerves two, three and four. Orthosympathetic fibers from T-11 to L-2 arrive via the hypogastric nerves and the hypogastric ganglia; the postganglionic fibers innervate the trigone, Bell's muscles and the crista urethrae; these nerves are relatively unimportant for micturition. The innervation of the bladder is seldom important in surgical procedures on the bladder itself but removal of the rectum, as in abdominoperineal procedures, may leave the bladder atonic by injury of its parasympathetic innervation.

The bladder wall is made up of a complex of interlacing, decussating muscle bundle tracts. The bladder neck is formed by the convergence of muscle tracts which sweep through to insert in the muscular wall of the urethra. The floor of the bladder is overlain by the muscular trigone, bounded by the two ureteral orifices and the vesical neck. This portion of the bladder is fixed and nondistensible.

Surgical Aspects of Bladder Anatomy

The surgical approach to the bladder is most often through the lower abdomen by either vertical or transverse incision. For all operations except those involving bladder tumor, the bladder is approached extraperitoneally by wiping upward the peritoneal fold overlying the anterior-superior aspect of the distended bladder and incising the exposed anterior bladder wall. This approach allows the drainage of urine or infected material without the risk of intraperitoneal soiling. Operations for bladder tumor usually require the preliminary opening of the peritoneal cavity to explore for metastatic lymph node or visceral involvement. In partial or simple cystectomy, the peritoneum is then closed and the bladder opened extraperitoneally. In radical cystectomy, the peritoneum overlying the bladder is best removed with the bladder, although this makes reconstruction of the pelvic peritoneal floor difficult or impossible. In any case, the peritoneum overlying a bladder tumor should always be removed along with the subjacent involved bladder wall.

Trauma to the abdomen will often rupture the distended bladder. The site of rupture may be extraperitoneal or intraperitoneal; since the distended dome of the bladder is the thinnest and weakest, rupture may take place in this area even though force was not directly applied at this point. The diagnosis is made by cystography, filling the bladder with a radiopaque contrast liquid. Extravasation of this material extraperitoneally will be revealed by a confined irregular extravasation within the pelvis. The free escape of contrast material into the peritoneal cavity with outlining of loops of bowel indicates an intraperitoneal rupture. Both types may exist and must be ruled out at time of exploration; surgical intervention is mandatory for repair of the ruptured area.

THE PELVIC URETER

As the ureter passes from the abdomen into the pelvis, it lies closely applied and loosely adherent to the undersurface of the posterior parietal peritoneum. Except in obese persons, it can usually be seen as it descends over the brim of the pelvis. The right ureter lies usually just medial to the cecum and the ileocecal junction and directly over the bifurcation of the right common iliac artery. On the left side, the ureter crosses the left common iliac artery slightly above its bifurcation but has a fairly constant relationship to the sigmoid colon, lying just beneath the intersigmoid fossa.

Pelvic Ureter in the Male (Fig. 4). After crossing the common iliac artery, the ureter turns outward and backward, lying below the hypogastric artery and following the curve of the sacrum. It then turns medially and forward to curve toward the bladder. Near its insertion in the superior portion of the fixed base of the bladder, the vas deferens passes medial to it and then hooks forward to approach the internal inguinal ring. The outer tip of the seminal vesicle is in close proximity at this point. The major arterial supply to the ureter near the bladder is derived from the inferior vesical artery. Other sources derive from the renal, internal spermatic, and the colic and middle rectal arteries. A plexus of veins surrounds the ureter near its insertion into the bladder.

Pelvic Ureter in the Female (Fig. 5). After crossing the common iliac artery, the ureter passes laterally, posteriorly and downward in an arch whose shape is the convexity of the pelvis. At the level of the ischial spines, the ureter turns medially and passes successively through the uterosacral ligament, Mackenrodt's (cardinal) ligament and the vesicouterine ligament.

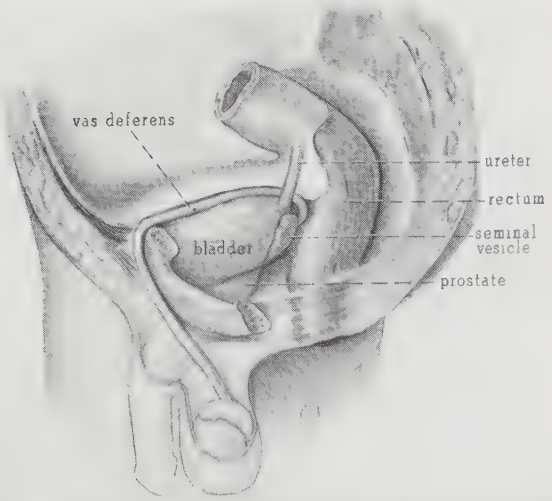

Figure 4. Pelvic ureter and ureterovesical junction in the male.

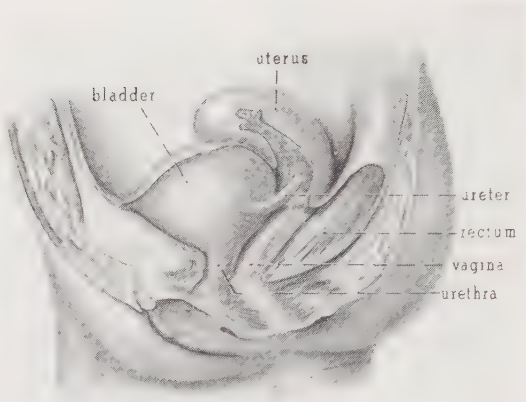

Figure 5. Pelvic ureter and ureterovesical junction in the female; the uterine ligaments have been cut away. Note proximity to vagina and cervix.

At this point the ureter is 1 to 1.5 cm. lateral to the cervix. It is intimately associated with the uterine artery during the first portion of its pelvic course and surrounded by a plexus of veins during its intermediate pelvic portion. The ureter then extends in the vesicouterine ligament to its insertion into the bladder, turning upward and medially to reach the bladder about 1.5 cm. below the level of the anterior lip of the cervix. It lies rather loosely in a canal composed of fibrous tissue as it passes through the vesicouterine and Mackenrodt's ligament. As the ureter pursues its pelvic course, the uterine artery lies lateral to its first portion, crosses its middle portion and lies medial to its terminal portion.

The nerve supply of the ureter is of little surgical consequence; the ureter can be completely denervated, as in a renal transplant, and ureteral function is unimpaired. Lymphatics of the pelvic ureter drain into the hypogastric lymph nodes.

Surgical Aspects of Ureteral Anatomy

Surgical injuries of the ureter occur most often in operations for removal of the uterus or rectosigmoid colon. The intimate relationship of the ureter to the uterine arteries and the distortion of surgical anatomy which can occur with uterine or ovarian lesions make hysterectomy the most common cause of injury. The preoperative insertion of ureteral catheters has saved many ureters from injury by making them palpably identifiable; it is undoubtedly true that many more ureters could have been saved had catheters been used.

The richly anastomosing blood supply of the ureter makes it possible to do many types of surgical procedures without fear of vascular embarrassment. This is particularly true if the ureteral adventitia is respected and preserved. Denuding the adventitia robs the ureter of the vascular supply which runs longitudinally in the adventitia.

Surgical exposure of the terminal half of the pelvic ureter is accomplished most readily through a midline vertical incision from pubis to umbilicus, the ureter being approached extraperitoneally. The proximal half of the pelvic portion may also be reached through this incision or by an oblique lower abdominal incision. The ureter is approached extraperitoneally; its adherence to the posterior aspect of the posterior parietal peritoneum brings it into view as the peritoneum is reflected medially.

THE URETEROVESICAL JUNCTION

The ureter pierces the bladder wall at the posterosuperior angle of the base on each side. Arterial and venous branches of the inferior vesical vessels overlie the ureter on its lateral aspect near its point of penetration. It passes downward and medially, piercing the bladder wall in an oblique tunnel approximately 1.5 cm. long in the adult. Current concepts hold that the ureteral muscle continues through to insert into the trigonal muscle, thus fixing the ureter to the trigone. The bladder wall is rather loosely applied around the intramural ureter. The combination of trigonal fixation with loose bladder wall investment allows the bladder wall to slide upward on the ureter as the bladder distends, thus making the course of the ureter more oblique during bladder distention and rendering the ureterovesical valve mechanism more competent by reason of compression of the ureter by increased intravesical pressure against the backing of bladder muscle. When detaching the ureter completely from the trigone as in an operation for reimplantation of the ureter, one is impressed with the ease with which, after circumscribing the trigonal stoma of the ureter, it is readily separated from the bladder wall throughout its intramural portion, being held only by the fibromuscular attachments of Waldeyer's sheath externally.

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Surgical Anatomy of the Pelvic Blood Vessels

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The peculiar angular relationship of the pelvic blood vessels, their closeness to the curve of the lumbar spine and sacral promontory with rich collateral supply to several organs, present situations which demand careful consideration by the general surgeon, urologist, neurosurgeon, and gynecologist, as well as the vascular surgeon.

All the arteries of the pelvis arise from the external iliac except the superior hemorrhoidal from the inferior mesenteric and the ovarian from the aorta.

The internal iliac artery is the largest artery in the true pelvis, arising as a terminal branch of the common iliac on the medial border of the psoas muscle. It passes posteriorly and downward across the medial surface of the psoas muscle and enters into intimate relationship with the ureter, its accompanying vein and the obturator nerve. (See Figs. 1 and 2.) It ends near the upper border of the greater sciatic notch by branching into an anterior and a posterior division which supply the inside and the outside of the pelvis.[11, 39]

ILIOLUMBAR ARTERY

One of the posterior branches is the iliolumbar artery which corresponds to what would be the fifth lumbar. It therefore passes upward and laterally to the iliac fossa. It is not accompanied by its vein in the pelvis. It has surgical application as pointed out by Mark, Lytton and Harvard of Yale,[20 21] who utilized it in cannulation of the hypogastric artery for chronic pelvic infusion of chemotherapeutic agents. (See Fig. 3.) It is ligated distally and cannulated retrograde to place the catheter in the first part

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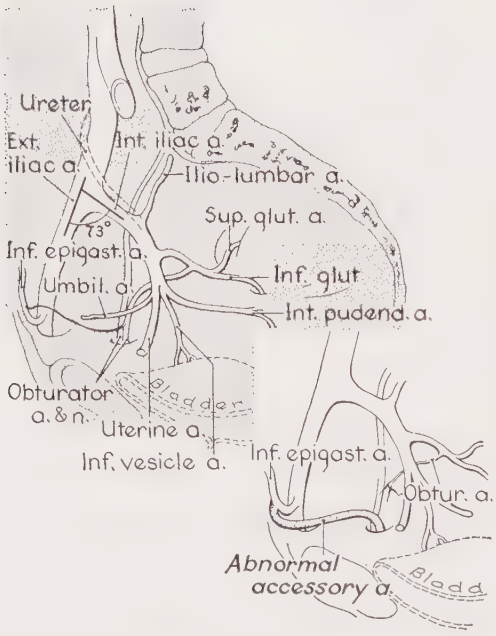

Figure 1. Sagittal view of the pelvic arterial supply. The angle between common and internal iliac artery is 73 degrees, much more than between common and external iliac artery. The abnormal obturator artery from the inferior epigastric is well developed. It can be seen that this would demand consideration in surgery of the incarcerated femoral hernia.

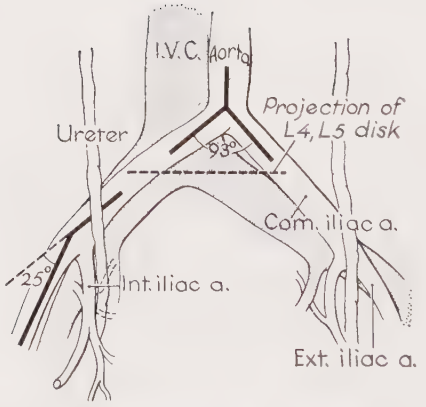

Figure 2. A front view of the pelvic vascular anatomy. The intimate relationship of the ureter, iliac artery and iliac vein is seen. It can be seen that the L_4-L_5 disc space is in proximity to the major venous channels. It can be observed that the blood stream must change its course by 25 degrees to flow from common to external iliac artery, but by 46.5 degrees to change from aorta to common iliac artery.

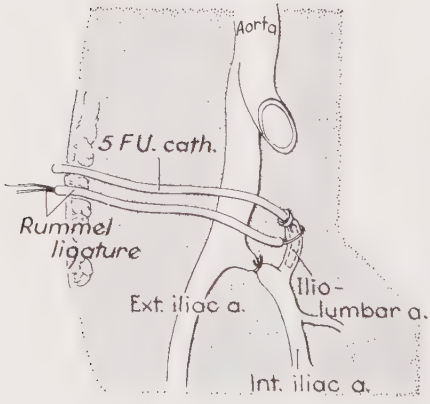

Figure 3. Method utilized by Lytton, Mark and Harvard, in cannulation of the hypogastric artery for chronic infusion of chemotherapeutic agents. The iliolumbar artery is controlled externally via the Rummel ligature. Hemostasis is obtained when the catheter is removed at the bedside.

of the internal iliac. This is held in place with a Rummel tourniquet led externally, and which can be used to achieve hemostasis after catheter removal at the bedside.

OBTURATOR ARTERY

The obturator artery passes downward along the lateral wall of the pelvis and emerges through the obturator foramen. An *abnormal* obturator artery is important in the surgery of femoral hernias. Pick, Anson and Ashley[27] have found the incidence of anomalous obturator artery to be high (47 per cent of cadavers had unilateral or bilateral anomalous obturator arteries). The inferior epigastric and the obturator each supply pubic branches, which anastomose on the back of the pubis (see Fig. 1). When the epigastric contribution is large, this is considered as one of the anomalous types (which occurs in 13 to 28 per cent of cases), and the situation is many times associated with, and may be attributed to, obliteration of the usual origin of the obturator artery. When this artery lies on the medial side of the femoral ring, near the lacunar ligament rather than lateral to the ring, it would almost completely encircle the neck of a femoral hernia sac.[15] This places the vessel in a precarious position since cutting this ligament in a strangulated femoral hernia may result in unexpected and sudden arterial hemorrhage.[39] Indeed, Pick, Anson and Ashley found that the anomalous artery passed through or ventral to the lipomatous tissue of the femoral ring (from which it is said femoral hernias may arise) in 38 of 43 anomalous cases examined.[27]

RUPTURE OF EPIGASTRIC VESSELS

Rupture of the inferior epigastric vessels and bleeding into the rectus sheath is a lesion which is not often seen, but which was described by Hippocrates.[2] Because it presents as an acute abdominal condition with peritoneal irritation, it can be quite confusing to the clinician if it is not thought of at the bedside.

Brodel has shown that the rectus muscle is long and narrow and has the ability to contract as much as 9 fingerbreadths. Terrific force can be generated during defecation, coughing or in raising the body from horizontal to sitting. The epigastric vessels are loosely attached to the muscle but closely adherent to the tendinous inscriptions.[5] With rapid and powerful contraction of the rectus muscle, Herrman has postulated that the vessels are unable to accommodate to the change in length and are torn loose.[16] It has happened relatively frequently in parachutists.[19]

The onset of exquisite pain is typically sudden and follows an act of violent contraction of the muscle. Progression depends on whether the

bleeding is arterial or venous. Signs include those of peritoneal irritation because the mass is usually below the line of Douglas and therefore bulges into the abdomen, stretching the poorly supported peritoneum. Systemic symptoms may be present and demand surgical intervention.[2, 16, 38]

UTERINE ARTERY

The uterine artery is a large vessel which usually arises from the anterior division of the internal iliac. It descends on the lateral pelvic wall in the area of the ovarian fossa where it is most safely isolated. It then passes horizontally toward the midline in the parametrial tissue where it crosses anterior to the ureter about 1 to 2 cm. from the lateral border of the cervix. It is at this point that the ureter may be injured during application of hemostatic forceps to ligate the uterine artery.[39]

UNILATERAL LEFT LOWER EXTREMITY EDEMA

The peculiar position of the bifurcation of the aorta and vena cava lying on the sacral promontory sets the stage for several clinically significant anatomic relationships. An interesting and perhaps controversial contribution to the subject of lymphedema praecox has recently been made by Calnan et al. [6] Lymphedema praecox[1] is described as occurring predominantly in females below age 40 and not present at birth. Its etiology has long been the subject of investigation and speculation.[18, 32, 40] No doubt hypoplasia, aplasia or malformation of lymphatic trunks is the cause in the majority of cases. It is interesting that the photographs and case reports in the literature indicate that the left leg vastly predominates in "unilateral and idiopathic" cases.[18, 32, 40]

Calnan et al. have demonstrated, by means of reduction in measured venous flow, by venogram and by surgery, four cases of left-sided lower extremity edema secondary to left iliac vein obstruction by the *right iliac artery*.[6]

Figure 4 represents the anatomy of a 12 year old girl showing that in

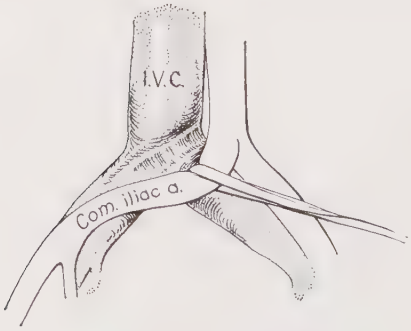

Figure 4. Illustration made at autopsy of 12 year old girl to show relationship of right iliac artery to left iliac vein. There appears to be a groove in the left iliac vein.

this normal specimen the right iliac artery actually does groove the left iliac vein. The right iliac artery is slightly taut as it curves around the convexity of the lumbosacral vertebra; and when the vein is injected with saline, it appears slightly compromised by the indentation of the artery.

It is also possible the abnormality may be in the fourth and fifth lumbar segments, which accentuate the compression of the artery. It is conceivable that lymphatic abnormalities are secondary to the venous obstruction in some cases. Treatment in Calnan's cases consisted of severance of the artery and rerouting it below the vein before resuture.

RELATION TO INTERVERTEBRAL DISC

At least 113 instances of vascular injury during surgical removal of an intervertebral disc have been reported.[35] Figures 2 and 5 show that the inferior vena cava and both common iliac arteries lie in surprisingly close proximity to the fourth lumbar intervertebral disc. This sets the stage for possible iatrogenic damage to venous structures during removal of a diseased intervertebral disc. Sudden and severe bleeding which can be inapparent and therefore not diagnosed would result if this relation were ignored.

LIGATION OF HYPOGASTRIC ARTERIES

Bilateral internal iliac artery ligation was first reported by Stephens of Vera Cruz in 1812.[37] Bier in 1893, proposed it as a preliminary step in radical pelvic surgery.[3] With improved technique such ligation has been largely abandoned, but in certain instances it remains an indicated procedure.

Hemorrhage still remains a cause of significant morbidity and even mortality among patients undergoing extensive pelvic surgery. Obstetricians know that postpartum hemorrhage ranks high as a cause of maternal

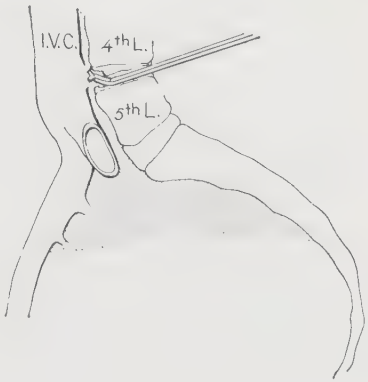

Figure 5. Shows the proximity of the L_4-L_5 interspace to the main venous channels. Inadvertent damage to the vena cava is possible during lumbar disc surgery.

mortality in the United States and that bleeding from the birth canal may not be controlled by hysterectomy because the vaginal artery quite often is not a branch of the uterine.[28, 31] Gynecologists have ligated the internal iliac artery with the ovarian for control of repeated bleeding from advanced cervical malignancy. This frees these patients from repeated hospitalizations because of hemorrhage.[8]

The control of bleeding during and after suprapubic prostatectomy is often difficult to accomplish. Kinman and Jennings[17] have performed bilateral hypogastric artery ligation as a prophylactic procedure prior to prostatic enucleation. It is beneficial to know that this procedure could be done to provide maximum hemostasis when extensive venous hemorrhage is a problem, for example, in abdominoperineal resection of the rectum.

The anatomic basis for the operation is that the blood supply to the uterus, vagina, bladder, prostate and perineum is primarily from the internal iliac vessels via the uterine, vaginal, vesical and pudendal arteries. On the other hand, sufficient collaterals are available to maintain viability of these organs.

The secondary blood supply to the pelvis is dependent upon anastomoses from the superior hemorrhoidal, ovarian, middle sacral, lumbar and inferior epigastric and other branches from the external iliac artery. Radiographs have shown these to be adequate.[34] Shafiroff et al. also experimentally ligated both internal iliac arteries of dogs. The supply from anastomotic channels was at first limited, blood pressure fell to 30 mm. Hg and became nonpulsatile. The pelvic viscera blanched, and cannulation of the distal segment yielded only 5 to 15 drops of blood per minute. This flow increased in 48 hours to 50 drops per minute. In two weeks the viscera again appeared pink, and pulsations were present distal to the occlusion. It is during the acute phase following ligation that the surgeon may capitalize on the reduced flow.[34]

An extraperitoneal or transabdominal approach may be used. Surgical consideration should be given to the relation of the hypogastric artery to adjacent structures. The superior hemorrhoidal artery lies in the sigmoid mesentery which is freed to expose the left iliac vessels. Immediately superficial to the artery and running parallel to it is an important structure, the ureter. Immediately deep to the artery and projecting from underneath on each side is the internal iliac vein. Laterally the obturator nerve is located in retroperitoneal fat. (See Figs. 1 and 2.) The transversalis fascia forms a sheath about the internal iliac vessels and blunt dissection can free the artery, but caution should be used to avoid laceration of the thin-walled vein. Ligatures should not crush the intima, as this area is prone to be involved by arteriosclerotic plaques which may be raised off the arterial wall by this maneuver. It must be mentioned that there is a small risk associated with this operation since there are three recorded instances of failure of collateralization with necrosis due to ischemia.[37] This complication was invariably of significance.

ARTERIOSCLEROSIS OF ILIAC ARTERIES

Until recently it has been somewhat of a mystery as to why different parts of the vasculature and indeed different segments of a given artery show striking differences in their degree of involvement by raised arteriosclerotic plaques. There are several anatomic facts which help explain this difference as it applies to the iliac vessels.

Schwartz and Mitchell,[33] reporting the incidence of raised arteriosclerotic plaques in autopsies, found that the hypogastric and common iliac arteries showed a high prevalence of disease, whereas the external iliac was remarkably free.

We know that a bend in a conduit (or vessel) creates turbulence. Szilagyi et al.[36] have demonstrated in canine preparations that an actual loss of flow rate occurs at bends, and have explained this on the basis of the laws of fluid dynamics, because of disturbance in the pressure head. Oil technologists know that when laminar or streamline flow becomes turbulent some kinetic energy is changed to heat energy and the pipeline becomes warm at the bends.

Rodbard and Johnson,[29] using an ingenious *in vitro* hydrodynamic preparation, examined the difference of laminar and turbulent flow on the deposition of flow-borne particles. They showed that an air stream bearing acid particles could deposit them onto susceptible areas of alkaline conduit walls. Phenophthalein was used as an indicator and showed that, where flow was laminar, deposition was minimal, but was increased under conditions of turbulent flow.

Schwartz and Mitchell suggest that the flow in the common and internal iliac arteries is turbulent because of their angle of origin from their parent trunks. This is borne out by the very close correlation between sites where turbulence is likely to occur and the prevalence of raised arteriosclerotic plaques.[33]

As can be seen in Figures 1 and 2 the three iliac vessels have varying angles of takeoff from their parent vessels. In the 12 year old female from which the drawing was made, it was necessary for the blood stream to change its course by 46.5 degrees to enter the common iliac, and 73 degrees to enter the hypogastric, whereas it was necessary to change only 25 degrees to flow to the external iliac artery.

On the other hand, the selective involvement of some arteries does *not* reflect differences in arterial wall structure. Schwartz and Mitchell explain, for example, that the common iliac artery is a muscular artery with only a slight dendritic ramification of elastic fibers. The external iliac shows similar histology but is quite dissimilar in its freedom from arteriosclerosis. The internal iliac contains significantly more elastic tissue but differs little from the common iliac in degree of atheromatous disease.[33]

It has been noted by some that many patients with aortoiliac occlusion (Leriche syndrome) are, quite peculiarly, young men between the ages of 30 and 50. DeWolfe et al. noted that 50 per cent of those with arterial oc-

clusion in the legs had diabetes mellitus whereas only 1 of 47 of their patients with Leriche syndrome had this systemic disease.[10] This suggests a local factor, and Gillman and Hathorn have furnished[12] experimental evidence to support local trauma as an etiologic agent. They showed that a single severe aortic injury to rats may be followed by arteriosclerosis in a number of months.

The abdominal aorta and the common iliac vessels, lying as they do between the ribs and pubis and on the unyielding anvil of the vertebral bodies, are subject to external trauma. (See Figs. 1 and 5.) Naide and Etzl have provided us with five clinical cases in which severe trauma (e.g., the abdomen of two had been run over by automobiles) preceded the aortoiliac occlusion by a number of years.[25] This report supports trauma as a local factor in the pathogenesis of Leriche syndrome.

RENAL TRANSPLANTATION

Experience gained by animal experimentation as well as observations on the human transplants thus far performed, has established a number of points that are related to the peculiar anatomy of pelvic blood vessels.

Ideally, the transplanted kidney would be placed in its normal anatomic position in the flank. This is rarely feasible because advanced renal disease causes a decrease in blood supply with a narrowing of the caliber of the renal artery.[13] Not only is it necessary to perform difficult ureteroureteral anastomoses, but it has been shown that hold-up of normal peristalsis occurs at the site of a ureteroureteral anastomosis.[26] Experience has shown that transfer to the right side of the pelvis of the recipient is the method of choice. Here the kidney lies in a relatively protected position in the iliac fossa and in an area with the same environmental temperature as the renal fossa.[13] Absence of the sigmoid and its mesentery on the right allows a better approach to the vessels. This position allows an ureterovesical anastomosis which is easier to make than the ureteroureteral juncture. This technique avoids infection and stricture which plague patients with the skin ureterostomy necessary in transplantation to the carotid or femoral circulation.[9]

Although the size of the renal artery approximates that of the hypogastric, the rich arterial anastomoses of the pelvic blood vessels allow the hypogastric to be ligated with relative impunity.[17, 28, 31, 37] In the event that the renal artery has several branches the early division of the hypogastric artery conveniently lends itself to accommodating multiple arterial connections.[23] (See Fig. 6.) This is an important consideration. Graves[14] has shown the segmental nature of renal arterial distribution (similar to the segmental arterial supply of the lung and liver) and he reinforces John Hunter's observation that the veins in the kidney "anastomose in large trunks but the arteries not at all." Segmental infarction would result from

failure to anastomose an accessory artery. Omission of aberrant venous connections is not critical.

In order to preserve the anterior posterior relationship of the artery and vein, the *left* (opposite) kidney is transplanted to the right iliac fossa. The left kidney is desirable also because of its long renal vein. The left renal vein must cross the aorta to reach the inferior vena cava whereas the right can enter much more directly. The right renal vein averages only 20 mm. in length while the left averages 57 mm.[30] In the right pelvis the vein must cross the external iliac artery to reach the interior iliac vein. Ross has shown that, when the left renal vasculature is unsuitable (too short a vein or multiple arteries), it is doubtful if it is worthwhile to explore the right side (in only one of 18 cases was it worthwhile). When the artery is anastomosed end to end, it is desirable to anastomose the vein end to side to achieve more stability and to gain an increased flow past the venous anastomoses. Should the internal iliac artery be too sclerotic to use, then the side of the external iliac may be anastomosed to the end of the renal artery.[23]

One of the interesting technical aspects of renal transplantation is that of fluid flow to the kidney. Hemodynamic studies are complex and variable since blood flow does not follow the mathematical equations of fluid mechanics applied to laminar and turbulent flow.[36] This necessitates more difficult *in vivo* measurements. Mims has shown, with a noncannulating electromagnetic flowmeter, a slight, greater decrease in the blood flow to the transplanted kidney with an end-to-side arterial anastomosis than with an end-to-end renal to hypogastric artery connection.[22] This can

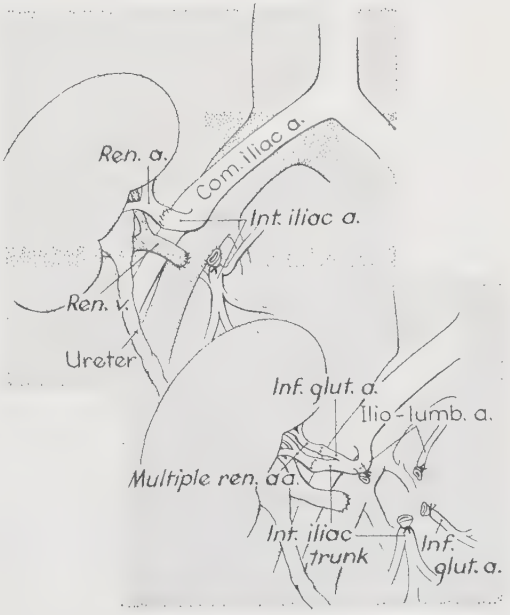

Figure 6. The hypogastric artery is anastomosed end-to-end with the renal artery in transplantation of the kidney. It can also be used to make connection with multiple renal arteries. The renal vein is anastomosed end-to-side.

be expected in the light of several anatomic and physiologic facts, now to be considered.

Szilagyi et al.[36] explained that a *bend* in a conduit causes turbulence and loss of flow rate because of the disturbance in the pressure head brought about by the abrupt change in direction of the flow. An increase in pressure head takes place along the outer wall of the bend and a decrease in the pressure head along the inner wall. According to the laws of fluid mechanics, the unbalanced centrifugal force of the fluid just inside the boundary produces a secondary flow outward along the plane of the bend, which is said to cause the fluid to move in a double spiral downstream. The loss of flow is proportional to the angle of bend. Furthermore, if fluid enters one conduit from another through a sharp-edged orifice, a converging of the stream lines takes place and turbulence is set up, dissipating part of the available energy as heat and therefore decreasing volume flow. If the orifice is more gradual, the loss of flow is less than if it is a slit. Therefore from a consideration of fluid mechanics, in an end-to-side anastomosis both the angle of union and the shape of the new orifice are theoretical factors affecting flow. Szilagyi and his associates have verified these theoretical considerations. In the canine iliac artery, the smaller the angle between the vessels, the greater is the flow. A bell-mouthed orifice created by excising a portion of the arterial wall markedly reduced the loss incident to construction of an end-to-side anastomosis through a simple incision in the iliac artery. For example, they found that *end-to-end* anastomosis gave a flow that was 97 per cent of normal, but that an *end-to-side* sharp-edged anastomosis could yield a flow as low as 60 per cent of normal, which increased as the angle between vessels decreased.

It has been shown that the denervated kidney placed in the pelvis retains its ability to undergo compensatory hypertrophy as well as to retain normal function in respect to concentrating ability, urea clearance, response to water load and excretion of phenolsulfonphthalein dye.[4, 24] It is able to remain free of infection.[24] The transplant, however, does have a tendency toward increased salt and water excretion.[9] Furthermore both the auto- and homotransplanted pelvic kidney have shown evidence of "autoregulation" of their blood flow. Cohn and Kountz,[7] using the square-wave electromagnetic flowmeter, determined the response of the renal artery, right femoral artery and transplanted renal artery to changes in arterial pressures. This was determined by graded constriction of the suprarenal aorta. Mean renal blood flow did not change in the pelvic kidney with a fall in systolic pressure until 70 to 80 mm. Hg was reached. On the other hand, both right and left femoral artery flow began to decrease relatively soon and paralleled the fall in mean arterial pressure. A pelvic kidney could maintain a flow which was comparable to that in the other normal kidney. This evidence suggests that the pelvis is not a low flow area and that the iliac vessels are quite adequate to supply blood to a transplanted kidney.

SUMMARY

The iliolumbar artery may be utilized to cannulate the hypogastric artery.

An abnormal obturator artery is important in surgery of an incarcerated femoral hernia.

"Spontaneous" hematoma of the rectus muscles has application to the diagnosis of the acute abdomen.

The relationship of the right iliac artery to the left iliac vein and the vertebrae may provide us with additional information about the etiology of unilateral left lower extremity edema.

Injury to the inferior vena cava or iliac veins may occur during intervertebral disc surgery.

Bilateral occlusion of the hypogastric arteries does offer important advantages in gynecologic, urologic or general surgery of the pelvis when there is danger of serious hemorrhage for which other means of control are not available.

The distribution of arteriosclerosis in the iliac arteries suggests a local factor. It is suggested that the varying angles of takeoff of the arteries are related to turbulence of blood flow and thus to the incidence of raised arterial plaques. Local severe trauma may play a part in the pathogenesis of Leriche's syndrome.

The pelvis provides an ideal site for the transplanted kidney. Here the ureter may be directly implanted into the urinary bladder. The hypogastric artery may be sacrificed with relative impunity, and the more efficient end-to-end anastomosis may be made even with multiple arteries. The left kidney is suited for transplantation to the right iliac fossa. Evidence shows that the pelvis is not a low flow area and that the kidney can function quite normally in this area.

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Surgical Anatomy of the Hand

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During the Renaissance Vesalius brought gross anatomy into focus. Since that time many investigators have embellished the basic structural studies with functional, physiological and philosophical observations. The forearm and hand have been prominently included in those observations. Sir Charles Bell[3] in his thought-provoking little volume, "The Hand, Its Mechanism and Vital Endowments as Evincing Design" (1837), presents a concept of hand anatomy placing it in proper context with man's position in the animal kingdom. Frederick Wood-Jones[33] probes more extensively into comparative anatomy and anthropology in his excellent work, "The Principles of Anatomy as Seen in the Hand" (1920). Duchenne[8] carried out detailed analysis of muscular function by isolated electrical stimulation. His classical volume, "Physiologie des Mouvements" (1867), was translated into English by Kaplan in 1959.

Allen B. Kanavel[10] published his monograph, "Infections of the Hand" (1925), in which detailed analysis of the spaces and synovial sheaths was made. "Surgery of the Hand" (1944) by Sterling Bunnell[4] became an indispensable reference during World War II. Recently Emanuel B. Kaplan[11] produced the nicely illustrated, detailed volume, "Functional and Surgical Anatomy of the Hand." Detailed studies of the integration of the intrinsic and extrinsic muscles operating the polyarticular digits may be found in the Studies by Stack,[25] Landsmeer,[12, 13, 14, 15] Kaplan,[11] Eyler and Markee,[9] Tubiana and Valentin,[31] and others. Discussion of the complicated mechanism is beyond the scope of this paper. The reader interested in more detailed study of functional hand anatomy is referred to the above listed volumes in addition to the papers by Littler and Chase[6, 16] whose broad experience in hand reconstruction has stimulated a practical concept of anatomy.

I shall draw heavily upon the writings of the authors above in delineating the following thoughts.

It is superfluous to eulogize the hand to surgeons, whose mastery of technical skills is so intimately related to integrated hand function under

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control of an alert central nervous system. The complicated motor balance at each joint resulting from contraction, fixation or relaxation of opposing muscle groups is worthless in the absence of the precisely fitted elements in the skeletal architecture. The skeletal framework with its restraining ligaments is beautifully designed but is useless without proper dynamic motor tension. The anatomical presence of both architecture and functioning muscles is only as serviceable as the integrity of the central nervous control mechanisms. As a unified interrelated melding of these elements emerges, hand function becomes the marvelous adaptable fact that it is in man. Its function is modified and further refined by sensory integrity. Sensation is protective and influential on central motor function. Moreover, special sensation as it resides in the hand makes the hand a special sense organ with which man explores his environment.

From the practical standpoint surgeons need to remind themselves of the anatomical basis for management of surgical problems in the hand. There are several unique anatomical facts that modify surgery in the hand.

SKIN COVER AND DEGREE OF MOTION

Without its normal mobile skin coverage the hand may be rendered useless. Scar fixation may be simple linear contracture or encasement of the part to the degree that all motion is destroyed. Before the surgeon makes an incision in the hand he must review hand motion in various planes and then place his incision in a manner that creates no limitation.

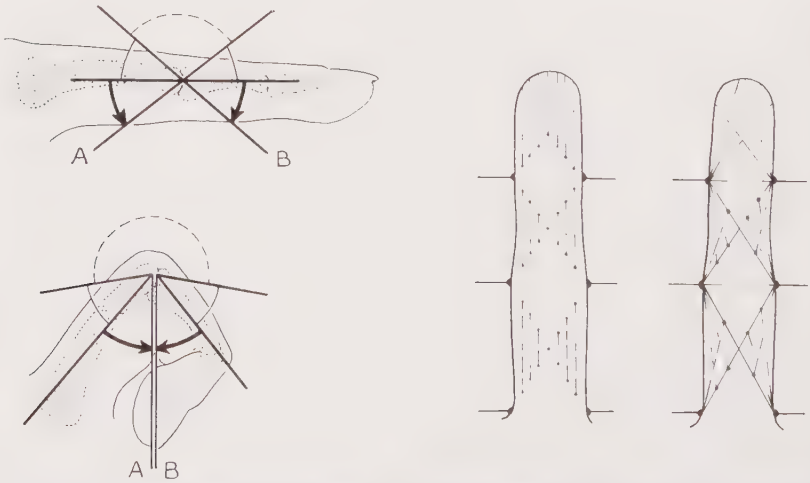

Figure 1. Schematic representation of the joint axes. The longitudinal dimensions in the midpalmar and mid-dorsal aspect of the digits change maximally. The midaxial line through the three joint axes does not change in length with flexion and extension. Palmar incisions placed longitudinally will produce contracture if they pass across the palmar diamonds delineated by lines joining the joint axes (after Littler). Transverse incisions are safe in avoiding flexion scar contractures.

Basically, incisions should be made in areas where motion does not alter their length. Geometrically each joint has a central axis around which it may move. The axis itself does not change and therefore this would appear to be a proper site for incision. The radii from this joint axis do not change in length with motion but the circumference does. Incisions along radii therefore are acceptable.

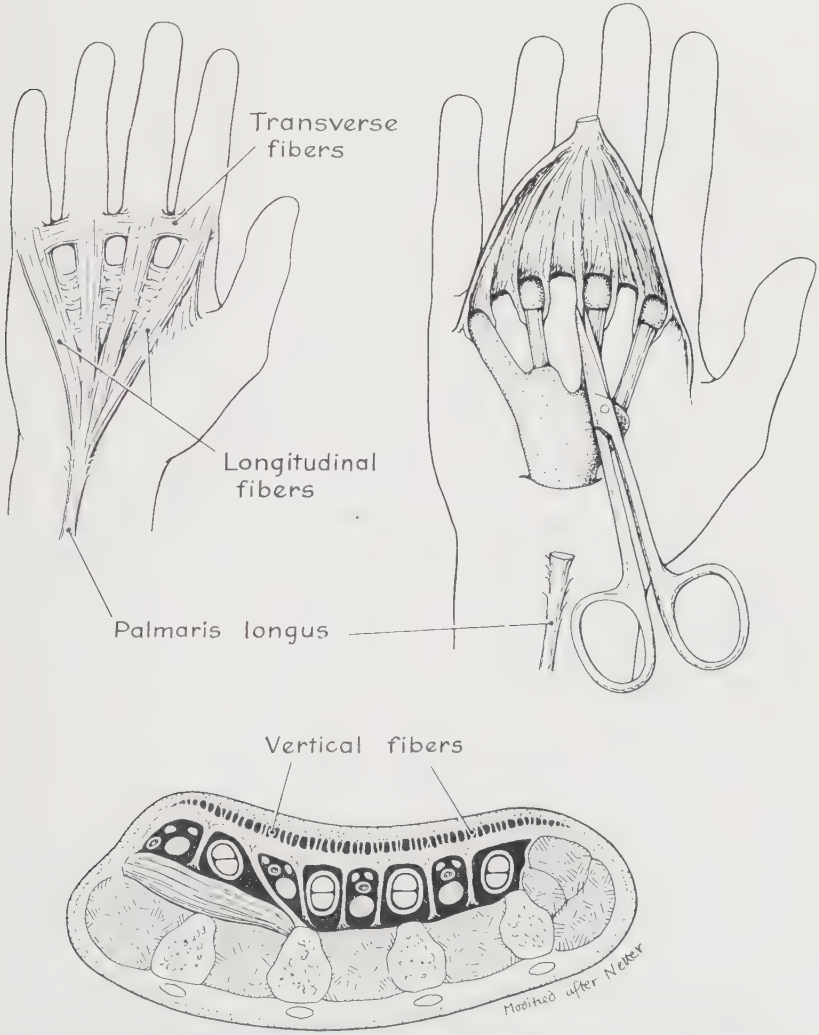

Figure 2. The palmar fascia with its longitudinal, transverse and vertical fibers. The longitudinal fibers with origin in the palmaris longus (when present) extend to the middle phalanx level. Transverse fibers are concentrated in the distal palm supporting the web skin. Vertical fibers extend superficial as multiple tiny tethering strands to stabilize the thick palmar skin. The deep vertical components concentrate in septa between the longitudinally oriented structures to the fingers. Removal of the fascia after dissection of skin from its superficial surface is best done by transecting the septa deep to the neurovascular bundles.

Any straight incision line that will intersect the joint axis point is acceptable. Any transverse incision in the fingers is also acceptable. (See Fig. 1.) The thick unyielding palmar skin may give rise to scar flexion contractures if longitudinal incisions are made in violation of this rule.

Dorsal skin by contrast does not give rise to contractures when incised. The obvious reason is an anatomical one. The loose areolar subcutaneous fascia allows compensation for local scar inelasticity by relaxation at a distance. Palmar hand skin is thick and fixed by the tiny skin attachments of the palmar fascia as well as Clelland's[33] ligaments. It cannot stretch from a distance to meet a need at the local area of contracture. Such skin fixation by anatomical structures allows little sliding of palmar skin. This is nature's answer to the need for stability of skin in grasping, pinching, and steadying objects with one's hand. It also protects the palm from avulsion injuries so easily sustained by the loose dorsal skin.

The palmar fascia, taking origin in the palmaris longus tendon, has longitudinal fibers which gather into separate flat bundles to each of the four digits. These fibers extend as far distal as the middle phalanges. Transverse fibers are concentrated in the distal palm where they support the soft skin in the interdigital web spaces. Vertical fibers are diffusely positioned as tiny tethering threads which attach to palmar skin. These tough attachments account for palmar skin immobility and for the fact that sharp dissection is required to separate skin from fascia.

The deep component of the vertical elements of the palmar fascia coalesce into septa which divide the longitudinally oriented structures to the digits into separate compartments. In removing the palmar fascia surgically the deep dissection is simplified by cutting the septa deep in the hand and, therefore, well away from the more superficial digital neurovascular bundles. (See Fig. 2.)

NEUROVASCULAR ORIENTATION

As transfer of composite elements of the hand on a neurovascular pedicle becomes more widespread, it is essential that not only the classical anatomy but also the usual variations be known. The recent study of arterial patterns in the hand by Coleman and Anson[7] is timely and informative.

The reader is referred to this paper for a detailed study of vascular patterns. The pattern of peripheral nerve distributions has been studied by Woodhall and Beebe,[34] who have pointed out the likely variations based on analysis of World War II nerve injuries.

In general, the divergent radial projection of the arteries from palmar arches is propitious as it allows movement of vascularized portions of digits through a wide arc to nearly any site in the hand. Similarly, the sensory nerve distribution from the median and ulnar nerves is arranged

as long branches from proximal palmar trunks, allowing facility for transfers. Parts of digits can be transferred on neurovascular pedicles because of the two neurovascular bundles to each digit. (See Fig. 3.)

When the superficial vascular arch is intact, receiving blood supply from both the ulnar and radial arteries, it may be transected at either end to allow adequate length in island pedicle transfers. Thus the whole arch may be reflected toward the recipient area.[23]

Note that the superficial arch crosses the palm at about the level of the fully abducted thumb.

Surgically, one may transfer composite parts with intact blood supply and sensory nerve supply on the neurovascular bundles alone (island pedicle).[17, 18, 19] In the hand which is dependent upon proper sensation for normal function, this technique is extremely important. A patch of skin and subcutaneous tissue may be moved from a nonessential part of the hand to the critical working surface of the prime digits (index digit or thumb) maintaining full spectrum sensation and intrinsic blood supply. (See Figs. 4 and 5.) This patch of skin has the unique advantage of bringing with it the specialized sensory end organs that are the basis for the hand's remarkable perceptive sensory acuity. Immediately after such a transfer, the patient continues to interpret touch on this patch as emanating from the donor digit. Some patients relearn quickly that the sensory patch resides on the recipient digit while others fail to achieve this central reorientation for months and years.

The technique of transfer of whole digits became well established and

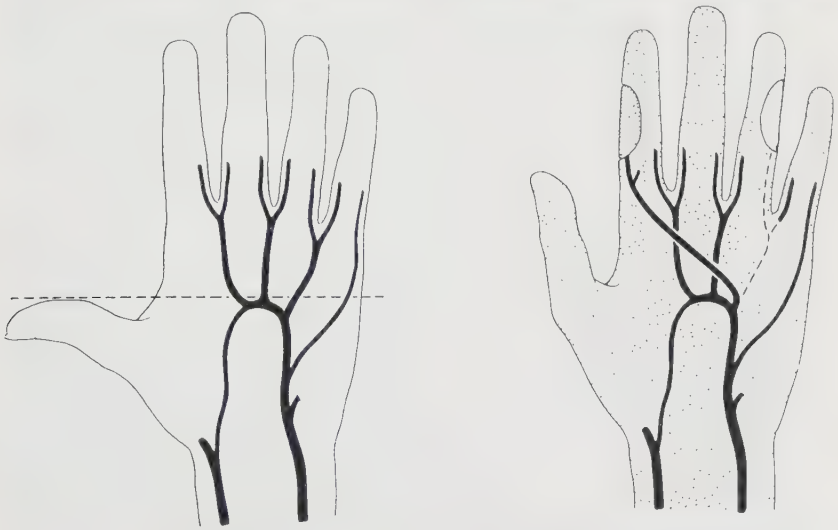

Figure 3. The superficial arterial arch and radial projection of the common and proper digital arteries are propitious since they make transfer of composite hand parts from one digit to another a practical reality. One may swing island flaps or whole digits through a wide arc from the superficial arch. Additional length may be secured by ligation and transection of the arch itself if it receives both radial and ulnar arterial perfusion.

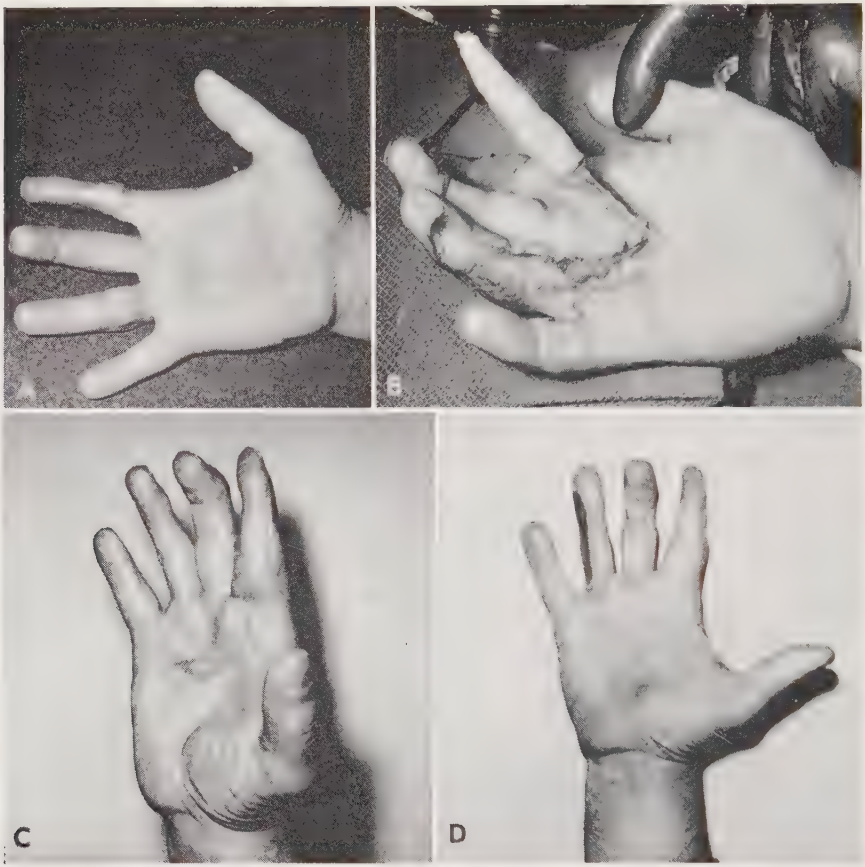

Figure 4. Severe avulsion injury of neurovascular structures and skin from the palm of the index finger (*A*). This was restored by island pedicle transfer of skin and soft tissue for the protected ulnar side of the ring finger (*B*). Radial orientation of nerves and vessels allows transfer across the hand to the working surface of the index digit (*C, D*).

predictable in modern hand surgery before the selective island pedicle transfer of skin and subcutaneous tissue alone. The arch and radial anatomy of the hand's vascular system and the longitudinal arborization of the peripheral nerves make these techniques a practical addition to the hand surgeon's versatility. Since World War II, thumb reconstruction has been more and more frequently done by transfer of the index digit on a neurovascular pedicle. Any of the four fingers may be utilized as a donor digit for thumb reconstruction but the many anatomical advantages offered by the index digit make it the best choice. The adjacent position, individuality of function, the established functional patterns with the thumb, size adequacy, and need for less flexor and extensor tendon reorientation are such advantages. In addition, the removal of the index ray guarantees a deep and adequate web space between the reconstructed thumb and the third digit.

Composite island pedicle transfers are very useful in utilizing damaged finger parts or nubs to lengthen the thumb. When digits on the radial side of the hand have been amputated together with a part of the thumb, an otherwise unimportant shortening of the thumb becomes very disabling. The foreshortened thumb fails to reach the ulnar digits and pinch function is lost. The additional length afforded by composite transfer of one of the remaining nubs becomes highly contributory in functional restoration. (See Fig. 6.)

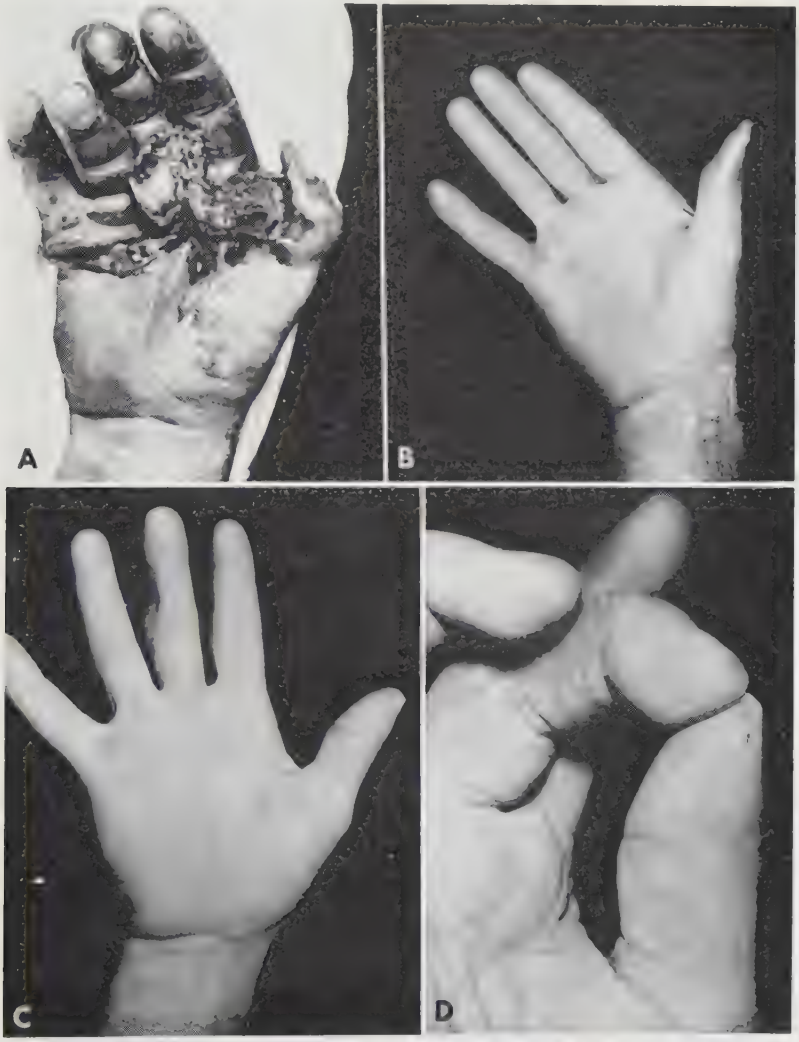

Figure 5. Severe avulsion injury of the thumb (*A*). Temporary skin cover afforded by grafted skin left an inadequate anesthetic thumb pulp (*B*). This was corrected by transfer of an island pedicle flap from the ulnar side of the long finger (*C*). Contour and sensory restoration was achieved (*D*).

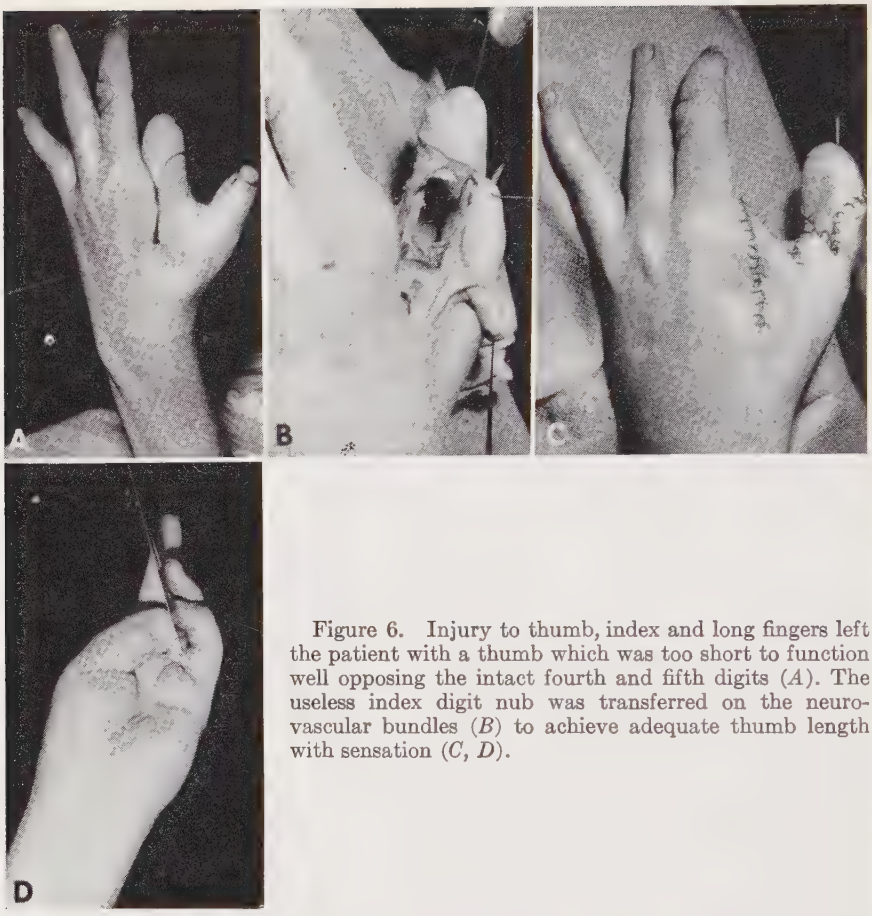

Figure 6. Injury to thumb, index and long fingers left the patient with a thumb which was too short to function well opposing the intact fourth and fifth digits (*A*). The useless index digit nub was transferred on the neurovascular bundles (*B*) to achieve adequate thumb length with sensation (*C*, *D*).

PERIPHERAL NERVES AND NERVE BLOCKS

The brachial plexus elements taking origin from the anterior primary divisions of $C_{5,\,6,\,7,\,8}$ and T_1 roots are anatomically handy for local anesthetic block in the neck and in the axilla. With the patient in a sitting posture with shoulder depressed and the head turned gently to the opposite side, it is possible to palpate the scalenus anticus just behind the sternocleidomastoid muscle. By digital pressure in the area adjacent to the palpable subclavian pulse one can commonly produce paresthesias in the arm and hand. It is nearly as easy to palpate the brachial plexus with the patient supine if traction toward the feet of the patient is applied to the relaxed arm. The clavicle is depressed, throwing the structures passing over the first rib into a subcutaneous palpable position. A short, small hypodermic needle is frequently all that is necessary to achieve paresthesias and proper local anesthetic injection.

The second site which is anatomically disposed for easy nerve block for the upper extremity is the axilla. With the axilla shaved and the arm abducted at the shoulder one can frequently see and certainly feel the axillary artery against the humerus. At the peripheral edge of the axilla where the vessel is easiest to feel just behind the biceps, the major peripheral nerves to the arm are all within the axillary sheath (anterior to the artery are median, musculocutaneous and medial antibrachial cutaneous; posterior to the artery are radial, ulnar and medial brachial cutaneous). A small-caliber, short hypodermic needle can be passed into the axillary sheath at this level using the artery as a landmark. There is a palpable resistance and release as the needle passes through the sheath. A deposit of local anesthesia anterior and posterior to the artery gives reliable regional anesthesia to the whole extremity and allows use of the pneumatic tourniquet. The patient may or may not experience paresthesias; but if the local agent is deposited in the axillary sheath, successful anesthesia is the rule.

At the elbow level the median and ulnar nerves can be blocked without difficulty. The median nerve is found quite constantly just medial to the biceps tendon and adjacent to the brachial artery. Using the artery as a guide, an effective block may be achieved by injection of 5 to 10 cc of anesthetic agent deep to the investing fascia at this site. The ulnar nerve in a subcutaneous position and somewhat protected by the medial epicondyle of the humerus is readily palpable. Oddly enough, it is sometimes difficult to achieve a good paresthesia and block of the nerve. With the elbow in relaxed flexion the best site for ulnar nerve block injection is just proximal to the medial epicondylar groove rather than in the groove. It must be remembered that the dorsal branch of the ulnar nerve comes from the main nerve at variable sites in the distal one-third to one-half of the forearm. To anesthetize the ulnar-innervated one and one-half digits, therefore, one should block the nerve at the elbow rather than at the wrist. Ulnar nerve block at the wrist must be accompanied by separate subcutaneous block of the dorsal branches if the fifth digit is to be truly anesthetic.

Wrist blocks of the median, ulnar, dorsal radial and dorsal ulnar nerves are very useful. The median nerve is the most superficial structure in the carpal tunnel passing beneath the flexor retinaculum. (See Fig. 8.) Paresthesias are readily obtained by inserting a small hypodermic needle either medial or lateral to the palmaris longus. As the needle penetrates the superficial fascia one may gently jiggle the needle and obtain paresthesias over the thumb, index, third and half of the fourth digit. Generally, inexperienced operators probe and probe too deeply within the forearm for this very superficial nerve.

The guide to the ulnar nerve is the flexor carpi ulnaris and the pisiform. The ulnar artery is palpable at this site. One injection just medial to the terminal palpable portion of the flexor carpi ulnaris will result in volar ulnar nerve anesthesia.

A general subcutaneous infiltration across the "anatomical snuff box" between the extensor pollicis longus and brevis tendons will effectively block the dorsal branch of the radial nerve to the hand. Similarly, a subcutaneous injection across the dorso-ulnar aspect of the wrist will block the dorsal branch of the ulnar nerve.

PERIPHERAL NERVES

The peripheral nerves to the upper extremity have anatomical relationships of great importance to the surgeon. The anatomical availability for nerve block has already been alluded to. Sites of particular susceptibility to injury coincide quite accurately with the sites chosen for anesthesia. The ulnar and median nerves are frequently injured just proximal to the wrist. The nerves are quite superficial and it is a region where injuries to all structures are frequent. Median palsy alone results in anesthesia over the important exploring and manipulating digits (thumb, index, long and part of ring) on the palmar surface. The median positioning muscles of the thumb become paralyzed, resulting in an inability to position the thumb for pulp-to-pulp apposition with other digits. In addition, the two radial lumbricales are paralyzed but this may be barely perceptible functionally.[11] Repair of the median nerve is best done primarily in clean lacerated wounds. There is a constant midvolar blood vessel on the median nerve that helps in its identity and in achieving very accurate axial rotation to perfect end-to-end apposition. The ulnar nerve is far less important from the standpoint of hand sensation but it is very important for its motor innervation of all the hypothenar muscles and interossei. In addition, it innervates the thumb adductor, deep head of the flexor pollicis brevis and the ulnar two lumbricales. *All* of the intrinsic muscles on the radial side of the flexor pollicis longus are median nerve innervated (abductor pollicis brevis, opponens, superficial head of flexor pollicis brevis). *All* other intrinsic muscles in the hand receive their innervation from the ulnar nerve. The tiny two radial lumbricales are the only exceptions to this axiom.

Essentially the flexor pollicis longus then divides the hand into a median and ulnar innervated part from the motor standpoint. The ulnar nerve divides into a deep motor branch and a superficial sensory branch just beyond the pisiform. Occasionally stab wounds of the hand result in transection of the motor branch with no sensory loss.

The ulnar nerve may be repaired primarily. A guide to proper funicular orientation is the fact that one can identify that portion destined to be the deep or superficial branches well above the wrist. The nerve may be bluntly split and funicular suture done for even greater accuracy.

Results from repair of peripheral nerves are equivalent if they are completed in the first two or three months after injury.[5] As the delay reaches six months, the results become less reliable. At the end of one

Figure 7. Late median nerve injury (*A*). The neuroma and proximal nerve scar were resected and the nerve was repaired. Since the nerve injury was more than one year prior to repair, the tendon transfer for paralyzed thenar muscles was done simultaneously with the nerve repair (*B*). (Courtesy of W. L. White, M.D.)

year, the results become quite poor. There are reported cases of repairs done several years after injury with successful functional restoration. Not only does the distal nerve sheath become less receptive to ingrowth of axones but the muscles undergo an irreversible denervation atrophy. Such atrophy can be delayed by post-denervation faradic stimulation with readily available portable battery-operated stimulators. If denervation atrophy of muscles reaches an irreversible stage but the nerve is still repaired to restore protective sensation, one may justify a simultaneous tendon transfer to substitute for the lost motor. (See Fig. 7.)

For detailed studies of the funicular pattern distribution and the intraneuronal plexuses the reader is referred to the extensive studies of Sunderland et al.[29, 30]

CROWDED AREAS WITH UNYIELDING BOUNDARIES

Flexion and extension motions in the hand occur at two levels, the wrist and the finger joints. Flexion at these joints would surely create volar bowstringing at the long flexor tendons unless it were prevented by the fixed retinacular conduits at these points. At the wrist the flexor retinaculum is the fixed roof of the carpal tunnel. The fibrous flexor sheath in the digits forms a fitted pulley system with condensed thickening in the areas of greatest fulcrum responsibility. At the wrist and in the digital fibrosynovial sheaths, anatomical structures create full occupancy. (See Fig. 8.) Any addition, be it more structures, post-injury or inflammatory swelling of contents, or inflammatory tightening of the sheath, sets the stage for inflammatory reaction and adhesion formation. Surgical repair of injured structures within these areas will result in unselected healing of the repaired structures to one another and to the fixed retinacular struc-

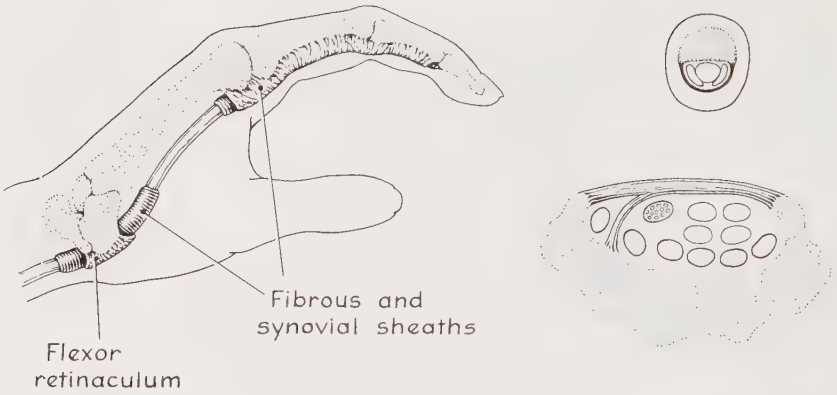

Figure 8. Crowded areas with unyielding boundaries occur where flexion mobility is greatest. The retinacular ligaments at the wrist and along the flexor aspect of the digit prevent bowstringing of tendons. These areas have little tolerance for further crowding by edema, repaired structures, or additional structures. Only selected structures should be repaired after traumatic transection at these critical zones.

tures. The prime function of tendons—that is, to transmit a muscle's power across the joints it influences—is destroyed by incarceration in such a wound. Considerations as to how to achieve repair without incarceration of the tendon have led to modification of the impulse to repair every structure transected. This is particularly so in the anatomically crowded pulley or fulcrum areas in the hand.

Experience of our mentors has shown that repair of a selected few anatomical structures can be tolerated with the expectation of retaining motion. Hence in transections at the wrist as in the digits, priority structures should be selected for repair. Other structures left unrepaired retract from the area leaving adequate space in these rigid tunnels for healing of priority structures. Thus, by circumventing compression and fixation, early mobility with the more probable restoration of a functional range of motion is assured. For details of management of structures transected at these critical levels the reader is referred to the well worked out concepts published by others (Peacock and Hartrampf,[24] Verdan[32]).

In general, if all flexor tendons are traumatically transected at the wrist level, only the distal phalangeal flexors are repaired (profundi and flexor pollicis longus). The superficialis tendons are left unrepaired leaving adequate space for edema and callus without compression and resulting massive adhesions. It is acceptable to repair the major nerves primarily or as an early secondary procedure. Injuries to flexor tendons within the digital theca are generally better left unrepaired primarily. There are obvious exceptions to this rule. Laceration of the profundus tendon within 1.5 cm. of its insertion may be repaired primarily by one of the many reinsertion techniques. Lacerations in children under eight years of age may be managed by primary accurate repair of the profundus tendons with predictable success. Under most other circumstances tendon graft

replacement of the profundus as a secondary procedure following lacerations within the digital theca gives best results.[24]

MOTOR RELATIONSHIPS OF IMPORTANCE

Extensors

The extensor digitorum to the fingers affords no independence of extension function to the four fingers because of its common muscle belly and the intertendinous bridges over the dorsum of the hand. The index and fifth digits have independent long extensor function by virtue of the extensor indicus and the extensor digiti minimi. (See Fig. 9.)

Incarceration of a single tendon of the extensor digitorum may checkrein extensor function in the other digits. The unacceptable technique of suturing extensor tendon ends over amputation stumps interferes with normal hand function.

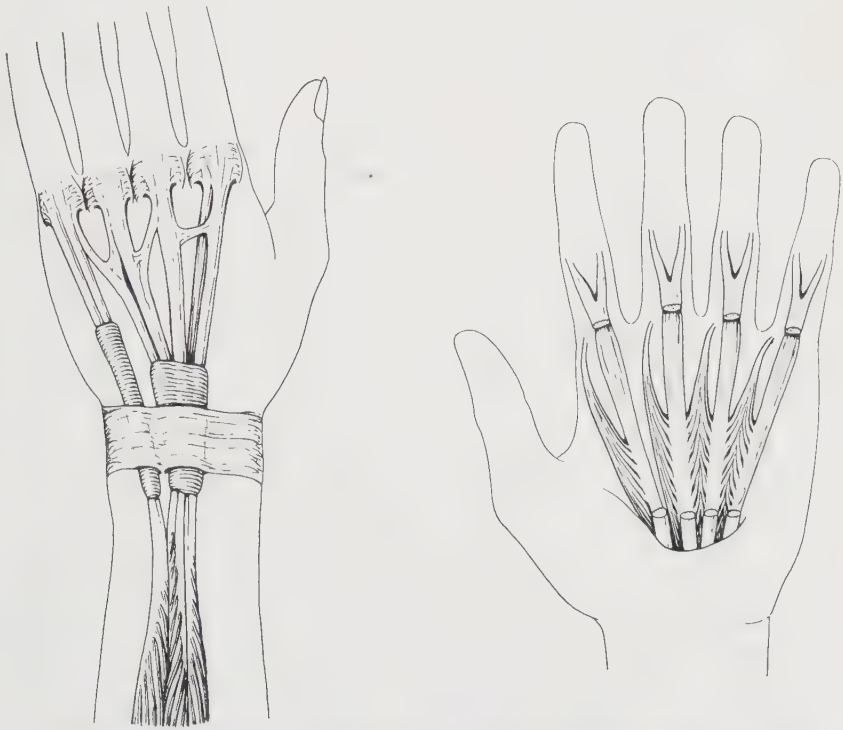

Figure 9. The extensor digitorum to all 4 digits lacks independence by virtue of a common muscle belly and the intertendinous bridges. Note how the extensor indicis and extensor digiti minimi (each on the ulnar side of the extensor digitorum to these digits) set the stage for independent index and little finger extension. The extensor digiti minimi is made up of two tendons—a fact of great importance when one uses this tendon as a transfer for another function.

Flexors

Anatomically the profundus tendons also lack independence of function. Independence of the index digit profundus, however, may be present to a varying degree. Whenever the profundus tendon is surgically fixed over an amputation area or advanced for reinsertion at its normal site, the surgeon must be aware of the resulting influence on the other fingers. He must also be aware of the moving origin of the lumbricales on the profundus tendons. Any shortening, lengthening or fixation of the profundus alters the balance in the total digital complex by changing the relationship of the lumbricale to the complicated extensor mechanism.

I would like to focus on the lumbricale as an example of a single muscle of great complexity and varying function. Actually, the action of the lumbricales was described by Galen in the second century A.D.

Backhouse and Catton[1] in 1954 made a study of the lumbricales, and they felt that they were important in interphalangeal extension.

Sunderland[28] in 1945 pointed out the importance of the lumbricales and interossei in extension at the interphalangeal joints. Sunderland made the point that it was the function of these intrinsic muscles to prevent hyperextension of the proximal phalanx by the extensor digitorum. This allows more efficient pull on the dorsal expansion (by the long extensor) extending the interphalangeal joints.

Backhouse and Catton,[1] as well as Long et al.,[20, 21, 22] noted on their electromyographic studies that the lumbricales are silent during total flexion of the entire finger but are very active whenever proximal or distal interphalangeal joints are extended actively or are held extended while the metacarpophalangeal joint is flexed actively. The lumbricales can be kept very quiet during metacarpophalangeal movements in any direction by keeping the interphalangeal joints fully flexed.

Kaplan[11] has made obversations on the activity of lumbricales during surgery under local anesthesia on humans. He noted a degree of contraction with the patient putting fingers through range of motion exercises and also by faradic stimulation and observation of the resultant digital posture. He has noted, as have many others, that paralysis of the radial two lumbricales causes little disturbance in the flexion of the metacarpophalangeal joints and extension of the interphalangeal joints of the index and middle fingers.

The profundus tendon has its primary action at the distal interphalangeal joint which it flexes. It secondarily flexes all other joints between its origin and insertion. If one fuses the distal joint, the profundus primarily flexes the proximal interphalangeal joint (becomes a sublimis). If the distal interphalangeal joint and the proximal interphalangeal joint are both fused, the profundus primarily flexes the metacarpophalangeal joint (like the intrinsics). If, instead of fusing the joints, they are fixed by the prime antagonist—the lumbricale—the profundus acts as a metacarpophalangeal joint flexor with interphalangeal joints extended.

Consider the lumbricale a controllable regulator of a loop around the

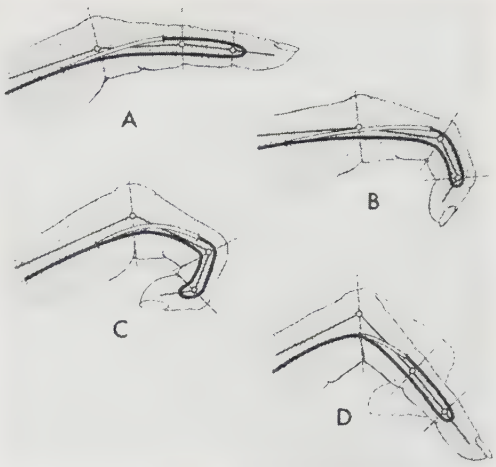

Figure 10. *A*, The lumbricale muscle is a moderator between the flexor and extensor mechanisms in the hand.

B, With the lumbricale relaxed the profundus pull flexes the distal joint primarily. The lumbricale origin moves proximally and its insertion moves distally putting the lumbricale on stretch.

C, If the lumbricale responds to the profundus pull which separates its origin and insertion, it may create metacarpophalangeal joint flexion in coordination with interphalangeal joint flexion.

D, Contraction of the lumbricale results in interphalangeal joint extension.

interphalangeal joints. The profundus tendon forms the palmar aspect of the loop and the extensor mechanism the dorsal aspect. The lumbricale closes the loop bridging across the flexor to the extensor side at the proximal phalanx. (See Fig. 10.) A pull on the profundus stretches the lumbricale by separating its origin and insertion. The origin is moved proximal by the contracting profundus. The insertion is moved distal as the distal phalanges flex under the profundus influence. As long as the lumbricale does not offer resistance, the distal, then middle phalanges flex with little influence on the metacarpophalangeal joints. If the lumbricale does not stretch but remains static, the profundus pull is transmitted to it and the metacarpophalangeal joint flexes. If the lumbricale actually contracts, the loop relationship changes and the interphalangeal joints extend, making the profundus primarily a flexor of the metacarpophalangeal joint through the lumbricale.

SYNERGISM AND ANTAGONISM

Synergistic and antagonistic groups of muscles in hand function must be considered when functional substitution by tendon transfers is contemplated. Obviously, certain groups of muscles have developed functional synergism which makes readjustment natural when one group is transferred to take on the function of the other. Examples of such natural synergism are the united functions of the wrist flexors and digital extensors or the wrist extensors and digital flexors.

Transfer of a wrist extensor to assume the function of a digital flexor requires little or no cerebral reorientation to function properly.

The importance of synergism in programming tendon transfer has been overemphasized in the past. The miraculous adaptability and capacity

for relearning and patterning of peripheral responses allow one to violate the general use of synergistic muscles in transfer substitution. Synergism is obviously the product of experience in movement. It is remarkable that man can learn to actuate separate muscle groups, separate single muscles, and even separate motor units within a muscle.[2] To achieve this one must have sensory feed-back from some source. Visual observation, peripheral sensory receptors and deep muscle-joint-tendon stretch receptors are most important sources for regulating musculotendinous function. Detailed studies of this important source of feed-back have been published by Kaplan.[11] Stilwell[27] has studied the innervation of deep structures of the hand in detail. He observes, "Several investigators have shown that central nervous connections are made and that qualitative and quantitative alterations in motor patterns can be evolved by proprioceptive reinforcement."

The *anesthetic* "blind hand" is clumsy by comparison with the normal hand. Such a hand is more dependent upon visual control alone which results in loss of dexterity. The addition of ocular blindness to extremity anesthesia essentially destroys fine function of the hand.

THE THUMB

The thumb complex has a loose and rather unstable skeletal architec-

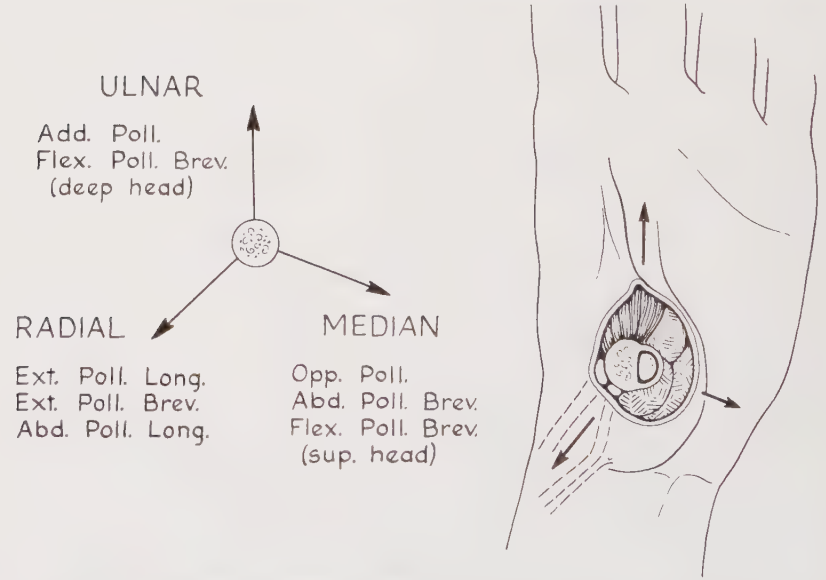

Figure 11. The first metacarpal relationship to the fixed carpal bones is dependent upon three groups of muscles. Each group receives separate innervation (median, ulnar and radial). Paralysis of any of these nerves results in imbalance of the forces stabilizing the thumb metacarpal. An abnormal thumb posture and functional deficit results which is classical for each nerve palsy.

ture. The carpometacarpal joint is a saddle joint with a lax capsule. This allows a wide range of circumduction motion and even a small degree of distraction of the joint on traction. Stability of the thumb root is heavily dependent upon the muscles affecting it. The radial innervated extensor pollicis longus and brevis and abductor pollicis longus secure the metacarpal dorsally. Opposing this to achieve stability are two groups of intrinsic muscles which, together with the extensor and dorsal abductor,

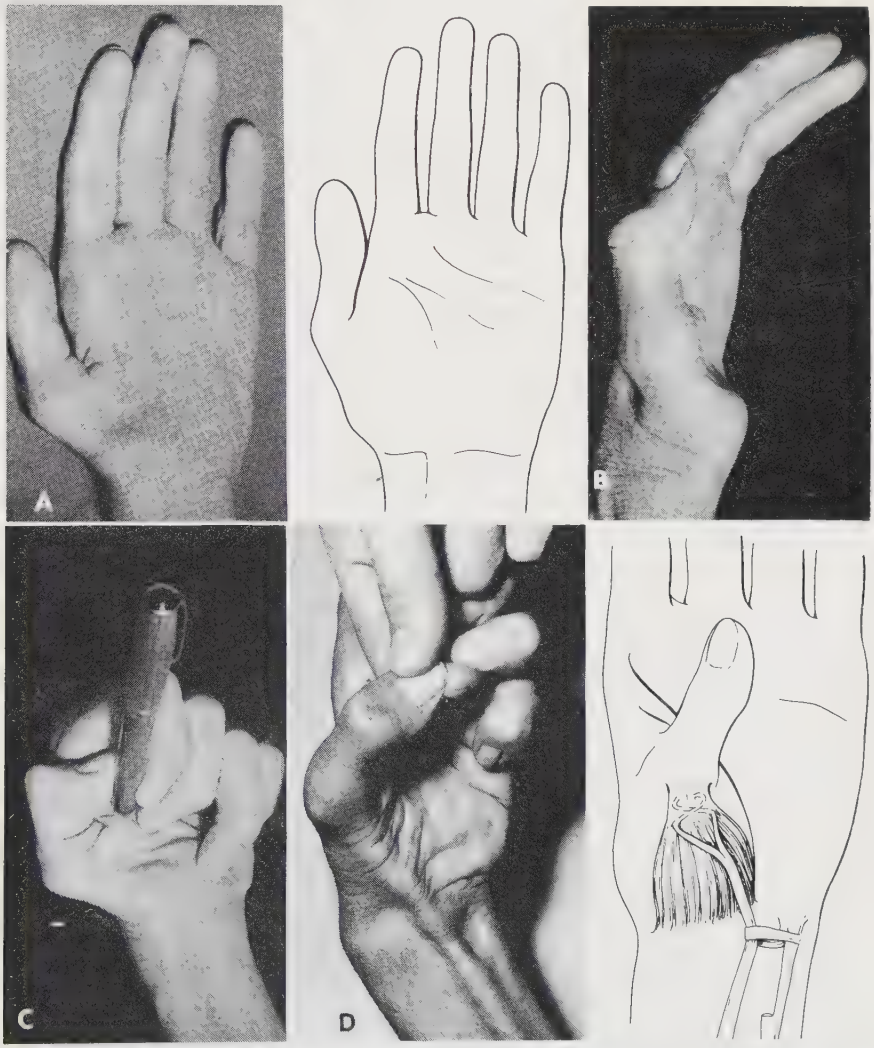

Figure 12. Median nerve palsy results in thenar atrophy (*A*, *B*) with resulting inability of the patient to achieve pulp-to-pulp opposition between the thumb and other digits. Note the abnormal pinch with lack of proper triangulation positioning of the first metacarpal between median, ulnar and radial innervated muscles by virtue of median palsy (*C*). The position was corrected by tendon transfer substitution for the abductor pollicis brevis using the fourth superficialis tendon (*D*).

triangulate the metacarpal. These two intrinsic groups are the median-innervated volar abductors (abductor pollicis brevis, opponens pollicis and superficial head of the flexor pollicis brevis) and the ulnar-innervated adductors (adductor pollicis, first dorsal interosseus and the deep head of the flexor pollicis brevis). (See Fig. 11.)

In the presence of paralysis of any of the three major motor nerves, thumb stability is compromised. In median palsy the positioning muscles of the thenar eminence are lost, resulting in inability to oppose the thumb for pulp-to-pulp opposition with other digits. (See Fig. 12.) Ulnar palsy results in adduction weakness and imbalance of structures influencing the metacarpophalangeal joint. Radial palsy destroys extension and dorsal abduction function with resulting adduction contracture which becomes fixed after an extended period of unopposed adduction.

Another characteristic of the thumb which makes it unique is that it has but two phalanges. Lack of a middle phalanx eliminates the need for two large flexor tendons; hence only the profundus counterpart exists (the flexor pollicis longus). The flexor pollicis longus has an independent muscle belly and independent function. This, together with the fact that it has no lumbricale originating from it, makes it functionally similar to the superficialis tendons. Its deep position in the forearm and distal phalangeal insertion make it like a profundus.

If a portion of the tendon is destroyed in the thumb, it may be advanced to the tip by inserting a tendon graft above the wrist. The absence of a lumbricale allows this without producing imbalance in total thumb function.

Motion in the metacarpophalangeal joint is quite variable from person to person. Its range is never very wide because of the strong collateral ligaments and strong capsule as well as by its surrounding muscles. Function of the thumb is diminished little by proper fusion of the metacarpophalangeal joint. When the joint loses stability by virtue of paralysis of the median or ulnar nerves or by paralysis of both, joint fusion results in dramatic improvement, especially when accompanied by opponens tendon transfer.

KINETICS OF THE UPPER EXTREMITY

The upper extremity is described as a "kinetic chain" of bones with a range of motion resulting from the freedom or limitation imposed by the sum of the ranges of joint motions from shoulder girdle to the finger tip.[26] In other words, although the interphalangeal joints are movable in one plane only as the total chain of upper extremity joints become involved, the digit has limitless possibilities for positioning in space. The metacarpophalangeal joint moves in two planes as does the wrist and so on up to the shoulder, which has tridimensional movement. Steindler[26] has reduced the

variations in motion possibilities to mathematical figures for freedom of motion, using the sum of degrees at each joint in the kinetic chain.

When motion created by various muscles in the upper extremity kinetic chain results in the same motion at the end piece, they can potentiate one another. For example, supination (as in turning a screwdriver) is attributed to the forearm supinators but it can be markedly potentiated by the major arm adductor at the shoulder. The full strength of these major muscles and the advantageous fulcrum create supination power of great magnitude.

The angle of application of a tendon influences its effect on the joint. As a joint angle changes, the angle of application changes and the pull becomes more effective. The angle of insertion may be increased by a pulley arrangement as by the condylar heads or by the sesamoid bones. The effect may be a variable one such as that of the sublimis on the profundus which pulls the sublimis into a more favorable angle of insertion as it straddles the profundus.

Deflection of muscles by the carpal groove is another example of pulley alterations of tendon function. The abductor pollicis longus, for example, is held by the pulley-like dorsal retinacular structures to direct its pull appropriately. This pull can be changed remarkably to become true oppositional pull by dislocation from the fixed tunnel and retinaculum.

There is a natural provision for muscle to become less powerful as it shortens by coincidental change toward the more favorable angle of insertion resulting in continuing strength at the area of influence.

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Varicose Veins

Anatomy, Re-evaluation of Trendelenburg Tests, and an Operative Procedure

R. STANTON SHERMAN, SR., M.D.*

Between 1928 and 1934, at the University of California Medical Center, patients were treated for varicose veins by injection of a sclerosing solution into the diseased vessels. It soon became apparent that this form of treatment was not satisfactory. High ligation was then instituted and in later years, was combined with retrograde injection of sclerosing solution. Over two hundred of these combined operations were performed. By 1940, although much improvement had resulted, it was evident that maximum benefits had still not been obtained. The presence of incompetent perforator veins in the thigh was believed to be the cause of this less than maximum improvement. Anatomic dissections were made on 137 thighs, and 465 patients (703 limbs) were surgically treated.[5] This work resolved many questions on the anatomy of the saphenous and perforator veins on the medial aspect of the thigh and knee and made it possible to locate perforator veins in the thigh. However, subsequent surgical procedures demonstrated that perforators in the thigh are only rarely incompetent. Anatomic dissections were then performed on 92 legs and surgical dissections were made on over 2000 additional patients.[6] It was found that incompetent perforator veins were very common in the lower leg and methods have been developed for eliminating these veins.

The present report reviews the anatomy of the venous system in the lower extremity, the principles of the tests to determine competency of that venous system, and an operative procedure to treat varicose veins.

ANATOMY

The superficial venous system of the lower extremity consists of the

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long and short saphenous veins. The *long saphenous vein* arises from the medial aspect of the foot and courses up the leg and thigh as two main tributaries. One of these lies superficial to the deep fascia throughout its entire length, whereas the other more important tributary lies beneath the deep fascia. This latter tributary courses proximally from the foot until it reaches a point approximating the junction of the upper and medial thirds of the thigh. Here it pierces the superficial layer of the deep fascia (fascia lata), joins with the superficial tributary, and proceeds upward as one vein to join the femoral vein in the fossa ovalis.

Figure 1 depicts the veins on the medial aspect of the thigh. At its upper end, the long saphenous B (union of deep [B′, B″] and superficial [B‴] veins after point C) vein receives a variable number of tributaries. Two important ones are the *lateral superficial femoral vein* (A) and the *medial superficial femoral vein* (W). This latter vein is important because it often has a tributary that makes direct connection with the short saphenous vein. It will be discussed later with the short saphenous. The lateral superficial femoral vein is often quite large and can be mistaken for the long saphenous at the saphrenofemoral junction.

At the proximal end of the long saphenous vein (B) just prior to its junction with the femoral vein, there are usually four tributaries: the *superficial circumflex iliac, the superficial epigastric, the superficial and deep pudendal veins.* Considerable variation may occur. For instance, the deep pudendal is often absent or it may connect with the superficial pudendal or the femoral vein. Often the superficial circumflex iliac joins the superficial epigastric to form a common trunk and this trunk in turn may join the long saphenous. Any one of these tributaries may join the femoral vein directly without having any connection with the long saphenous vein. Thus, it is imperative that the femoral vein be widely exposed during surgical ligation so that no tributary will be missed.

The fascia lata of the thigh continues without interruption down the leg to the base of the toes as a distinct enveloping sheath, separate from and overlying the deeper (crural) fascia of the lower leg. As stated above, the more important of the tributaries of the long saphenous lies under this enveloping sheath and extends from the foot to about the junction of the upper and middle thirds of the thigh. The point of emergence (C) from beneath the fascia lata varies from 13.5 to 33 cm. above the upper edge of the patella, with an average distance of 20.37 cm. This emergence at C occurred in all of 101 anatomic dissections.[5]

Any perforating vein that makes connections between the femoral vein and the long saphenous on the medial aspect of the thigh does so through the deep saphenous tributary (B′, B″). In 56 consecutive anatomic dissections, the *mid-Hunter vein* (MH) was found to do so in all cases.[5] A constant tributary of the mid-Hunter perforator is the *subsartorial vein* (SM) which drains the sartorius muscle. This vein courses distally, sometimes ending in the sartorius muscle but in some manner, either directly or

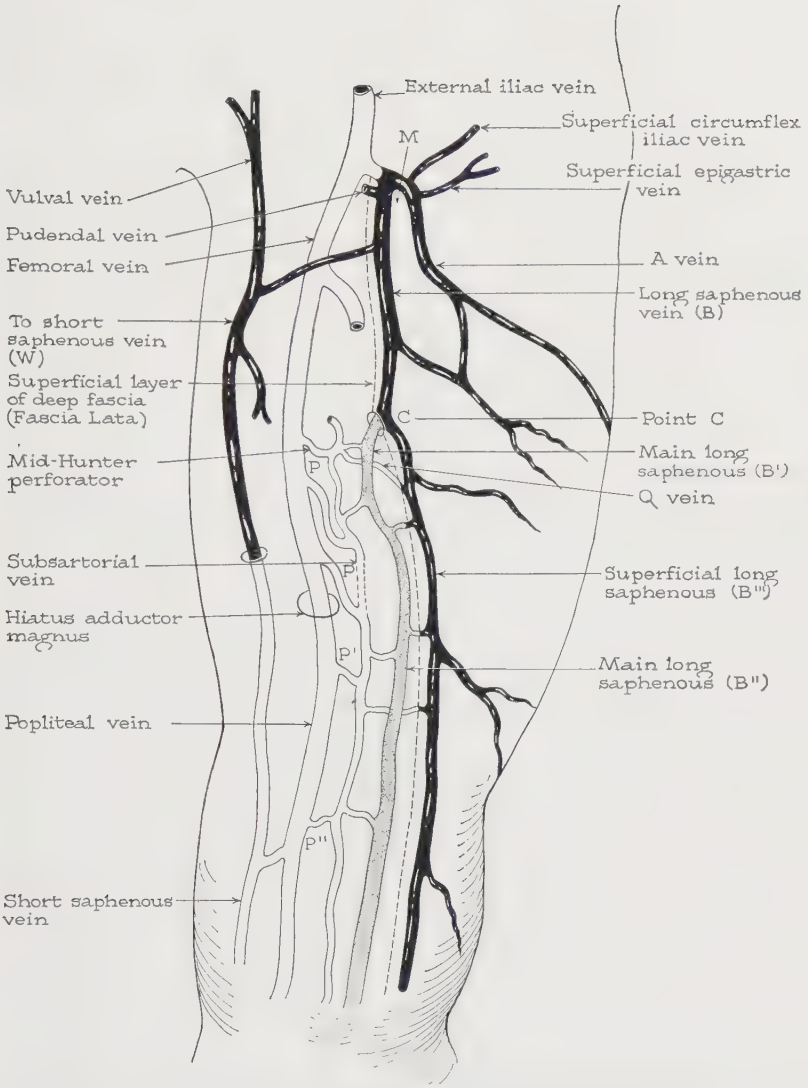

Figure 1. Drawing of medial aspect of thigh showing the saphenous vein as A and B. B′, B″, B‴. Veins A, B and B‴ are superficial to the deep fascia (fascia lata) whereas veins B′, B″ lie beneath the superficial layer of the fascia lata. The constant mid-Hunter canal perforator connects the femoral vein with the main saphenous B′. The genicular venous plexus (P, P′, P″) connects the femoral and popliteal veins with the subsartorial vein. The subsartorial connects with the main long saphenous (B″) in the thigh, and continuing distally in the leg, makes connections with B″, B‴ and the posterior tibial vein.

Accessory perforator vein Q merges and connects with the superficial saphenous B‴ without making direct connections with the main saphenous B′, B″.

Line M illustrates the possibility of mistaking superficial vein A for the main long saphenous when two saphenous veins are present.

through the sartorius muscle, makes connection with the genicular plexus (P, P′, P″).

The importance of the subsartorial vein is that it gives rise to other perforator tributaries that connect with the deep saphenous (B′, B″). The genicular plexus also is responsible for perforators between the deeper saphenous (B″) and the distal femoral or popliteal veins. Although vein P P′ is a genicular plexus vein, it can be considered to be a distal extension of the subsartorial vein (SM). It continues distally into the lower leg to make connections with the highest perforator. In addition, there may be accessory perforator veins (Q) that arise from the mid-Hunter (MH), subsartorial (SM), or genicular plexus (P, P′, P″) veins and, after piercing the deep fascia, join with the superficial saphenous tributaries without making direct union with the deep saphenous vein (B′, B″). Simple stripping of vein B′, B″ would not eliminate such perforators.

The *deeper saphenous vein* (B″) lies adjacent to the medial border of the tibia in the area just below the knee while in the lower part of the leg it varies from 1 to 2 cm. posterior to this border. The vein B″ unites with the deep veins by perforators and with the superficial saphenous vein (B‴) by vessels that pierce the fascial layer (crural). The placement of the perforator veins in the legs has been previously published.[6]

There are six perforator veins on the medial aspect of the lower leg (Fig. 2). Perforators 3, 4 and 5 usually make direct connections between the main deep long saphenous vein (B″) and the posterior tibial vein. Perforator No. 1 is about 2 cm. posterior to the medial border of the tibia and perforator No. 2 is approximately 1 cm. posterior to this border. These two perforators seldom make direct connections with the posterior tibial vein, instead they connect with secondary veins which in turn join with the long saphenous vein (B″) as well as with other superficial veins (B‴). Perforator No. 6, which is 1 cm. medial to the medial border of the tibia, makes connections between the main long saphenous B″ and superficial veins B‴ as well as with the posterior tibial vein and the genicular vein P, P′ (an extension of the subsartorial vein). The placement of these six perforator veins is quite constant, especially that of No. 2. Beginning with No. 1, they are about 13.5, 18.5, 24, 30, 35 and 40 cm. above the sole of the foot.

There are two longitudinal lines of perforators on the lateral aspect of the leg (Fig. 3). The first lies in the intermuscular septum between the peroneus longus and brevis muscles on the one hand and the gastrocnemius and soleus muscles on the other hand. These perforators make connections between the peroneal vein and the superficial saphenous veins (B‴) and occur at about 5-cm. intervals between 10 cm. and 45 cm. above the sole of the foot. Nearly all incompetent perforators on the lateral aspect of the leg are found in this longitudinal line.

The second line of perforators lies between the peroneus longus and brevis muscles posteriorly and the tibialis anticus anteriorly. Usually there

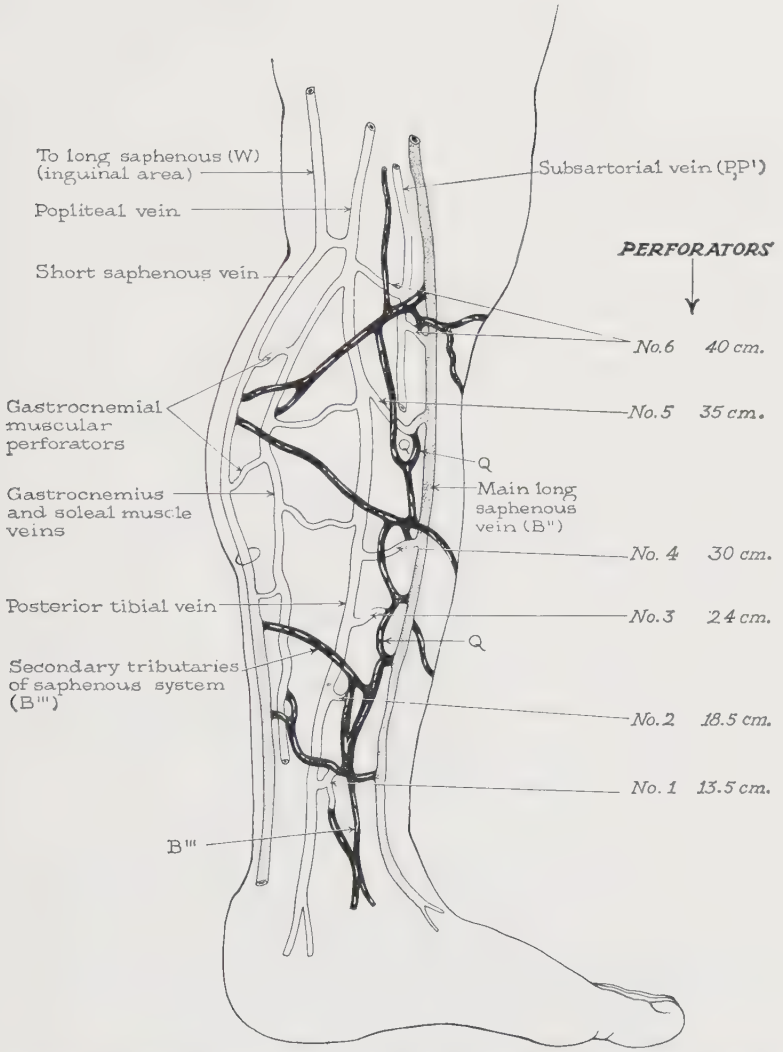

Figure 2. Drawing of medial aspect of leg showing the saphenous system (B'', B''') and the short saphenous vein. The white veins are situated beneath the crural fascia. The stippled veins (B'' and the lower part of the short saphenous) lie between the extension of the deep fascia of the thigh and the crural fascia. The superficial saphenous tributaries are black.

Perforator veins 1 and 2 connect the posterior tibial vein with secondary saphenous veins (B'''). Perforator No. 6 connects the posterior tibial and subsartorial P, P' with the veins B'' and B'''. Perforators 3, 4 and 5 connect the posterior tibial vein with the main long saphenous vein (B'').

The accessory perforator vein Q emerges from beneath the deep fascia without making direct connections with vein B''. All measurements start from the sole of the foot.

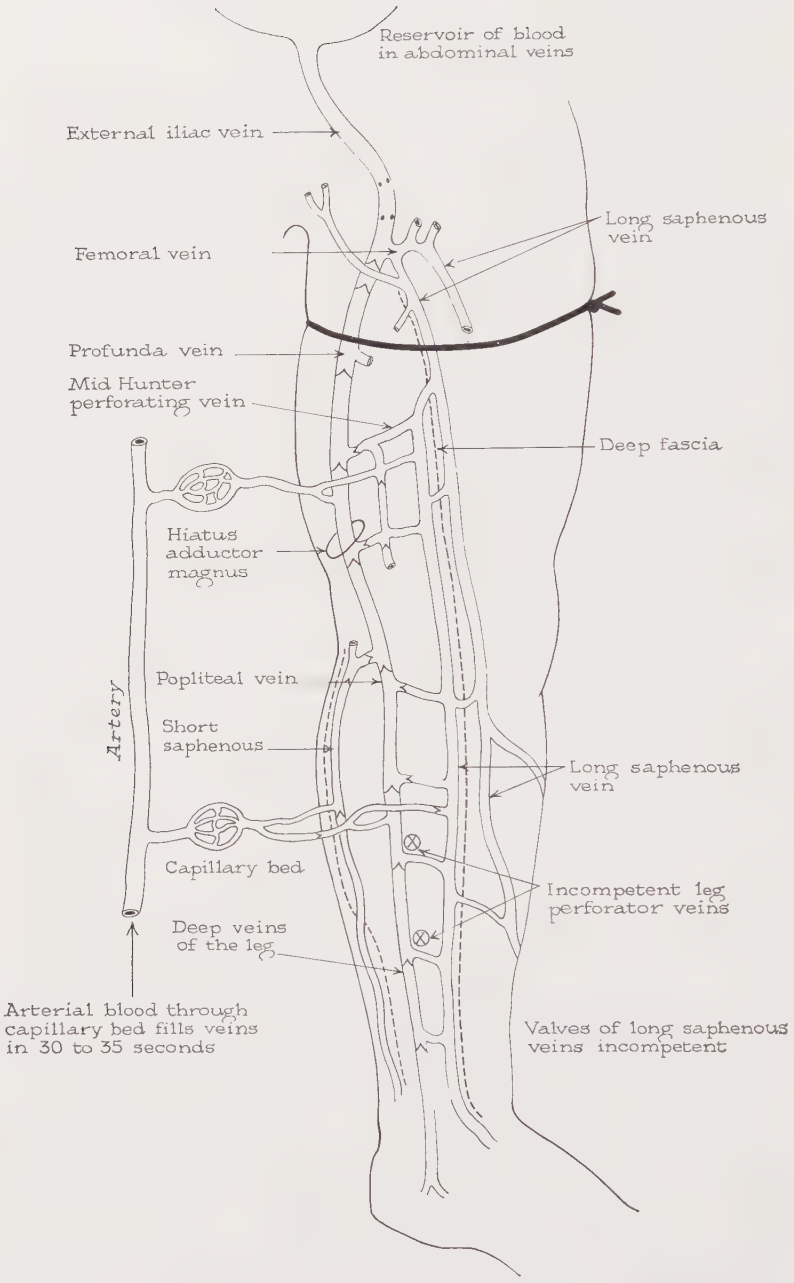

Figure 3. Schematic drawing indicating the veins and arteries of the lower extremity of a patient possessing varicose veins. The blood is pumped by the heart into the limb and, after passing through the capillary bed, it fills the previously emptied veins. Although the number of valves in the external iliac vein varies from none to three, two valves are shown to be incompetent. No valves are shown in the saphenous system in order to indicate that they are incompetent.

are about six of these perforators which also occur about 5 cm. apart. In patients with simple primary varicose veins, incompetent perforators are rarely found in this area.

In the calf of the leg, there are venous connections between the superficial veins B''' and the short saphenous on one hand, and the veins within the soleus and gastrocnemius muscles on the other hand (Fig. 2). Only a few of these make connection with the short saphenous; most make connection with the *superficial veins* B''' which are tributaries of both the long and short saphenous veins. These superficial veins B''' pierce the crural fascia and join the veins within the muscles. These veins from the various muscles finally unite with the posterior tibial vein at various levels, the highest joining to the popliteal vein approximately 1 cm. below the saphenopopliteal junction. As a generalization, there are four longitudinal lines of perforators on the posterior aspect of the leg. Two of these are about 2 cm. and the other two, 3 or more cm. on either side of the midline. There are about four perforators in each of the three medial lines and only one or two in the most lateral line. The lowest perforators are 10 cm. and the highest 45 cm. above the sole of the foot. In the lower leg as in the thigh there are accessory perforator veins (Q) that make connections between the deep veins and the superficial veins (B''') without making direct connections with the main saphenous (B'').

The perforators of the foot will not be discussed because they seldom cause difficulties in patients with plain varicose veins. For reference, they are detailed in a previous article.[6]

The *short saphenous vein* arises at the lateral border of the foot, posterior to the external malleolus and courses proximally to the popliteal

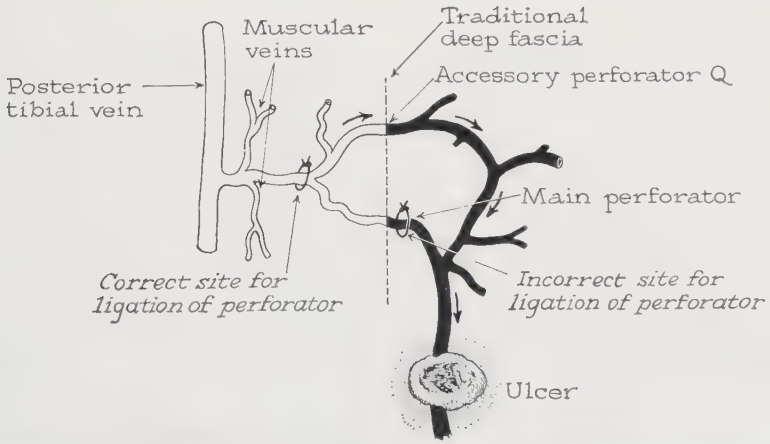

Figure 4. Illustrating the method of ligating an incompetent perforator vein. Two perforators are shown as emerging through two separate fascial openings. If ligation is made at the indicated incorrect site, blood will reflux through the accessory perforator Q vein, as indicated by arrows. Ligation must be made deep beneath the crural fascia at the point designated as the correct site.

fossa (Fig. 2). The lower half of this vein lies rather superficially between the deep (crural) fascia and the extension of the superficial layer of the deep fascia of the thigh (fascia lata). In this respect it is similar to the main long saphenous (B″). At about the middle of the lower leg, the short saphenous pierces the deep (crural) fascia and proceeds proximally to the popliteal fossa. The short saphenous usually makes a connection with the popliteal vein about 2 cm. above the popliteal skin crease behind the knee, although it may connect as high as 10 cm. above the skin crease. There are many other possible variations: for instance, there may be two or three connections between the short saphenous or its tributaries and the popliteal or other deep popliteal fossa veins. One such connecting vein may be located at the lateral canthus of the popliteal skin crease. Or, the short saphenous may make only trivial (if any) connection with veins in the popliteal fossa. Instead, the "W" tributary of the short saphenous (Figs. 1 and 2) remains between the fascial layers and courses proximally, first on the posterior and then on the medial aspect of the thigh, to join the long saphenous vein several centimeters distal to the saphenofemoral junction. This extension of the short saphenous also pierces the deep fascia at about the level of the midthigh to occupy a position superficial to the deep fascia. Again the short saphenous vein, instead of joining the popliteal vein after entering the popliteal fossa, often makes connections with other deep veins, such as the veins draining the medial or lateral hamstring muscles or tributaries of the profunda femoris. The short saphenous may, without any connections with the popliteal fossa veins, join the long saphenous vein just below or above the knee joint. Rarely, the short saphenous ends abruptly in the midcalf and joins with a vein in the gastrocnemius muscle.

TESTS

Tourniquet tests employed in locating sites of incompetent venous valves in the lower extremity are detailed in many articles. Because the interpretation of these tests has been so confusing that many clinicians refuse to use them, a re-evaluation is in order. The simple Brodie-Trendelenburg test will be used as an example, for with the exception of the walking and Schwartz tests, the same interpretation applies to all.

Figure 3 is a schematic drawing of the veins of the lower extremity and the placement of the venous valves. Also indicated are the only two sources of blood capable of filling previously emptied veins in the leg. The first source is the huge reservoir of blood in the valveless abdominal veins; the second source is the combined arterial and capillary beds. Basic to all tests that determine competency is the draining of all venous blood from the limb and measurement of the time required to refill the vessels.

When employing the Brodie-Trendelenburg test, the patient is placed

in a supine position and the leg is elevated. This maneuver is simulated by holding Figure 3 upside down. It is obvious that all the venous blood in the deep and in the superficial veins of the leg will be emptied into the abdominal reservoir. A tourniquet is then applied around the thigh a little below the saphenofemoral junction. The patient then assumes the erect position (Figure 3 should be right side up) and the tourniquet is immediately released. This procedure is repeated as before except that the tourniquet is left in place for 30 or more seconds.

The only available blood that can precipitately refill the veins in the leg is that in the abdominal reservoir. If the valves in the external iliac, proximal femoral and saphenous veins are incompetent, a sudden filling of the leg veins occurs when the tourniquet is released.

Another source of blood is from the arteries and the capillary beds. In normal persons, the veins in the leg refill in ten seconds regardless of whether the tourniquet is on or off.[1] However, in patients with varicose veins, if the tourniquet is left on, the time required for refilling is much longer, 30 or more seconds, and it varies considerably depending on the overall capacity of the varicose veins.

The Brodie-Trendelenburg test is termed positive if the veins fill at once when the tourniquet is released immediately on standing or if they fill slowly and are not entirely full at the end of 30 seconds when the tourniquet is not removed. The usual interpretation of this positive test is that the valves of the long saphenous are incompetent and that the valves of the perforating veins are competent.[1] My interpretation agrees that the valves of the long saphenous are incompetent, and I believe that any valves in the external iliac and femoral vein proximal to the saphenofemoral junction must also be incompetent. If these latter valves are competent, it will be impossible to have a sudden filling of the leg veins from the abdominal reservoir. The second part of the standard interpretation of the positive Brodie-Trendelenburg test (that the perforating veins are competent) is open to criticism. If we look again at Figure 3, we see that the arteries and the capillary beds can be constantly filling the venous system. Normal veins become filled in ten seconds, but larger varicose veins with much greater capacity require 30 or more seconds for refilling. Even if perforators with competent valves are present, they will have no access to the abdominal reservoir of blood if the valves in the femoral vein below the saphenofemoral junction and in the profunda veins are proved to be competent. And as the limb is completely emptied of venous blood, all that occurs is that the leg veins are gradually being filled from the arteries and capillary beds and the positive test gives no information whatsoever concerning the competency of the perforator veins.

If, in the test described, the veins in the leg fill slowly when the tourniquet is not removed and are entirely filled at the end of 35 seconds, and if they do not fill any faster if the tourniquet is released immediately, the test is said to be negative. It is agreed that the usual interpretation of

competency of the long saphenous is correct. However the interpretation of incompetency of the perforators is without foundation. The veins are simply being filled with blood from the arterial tree. No information is obtained concerning the condition of the valves in the perforator veins.

When the varicose veins fill at once in both parts of the test, the test is said to be double positive. The interpretation that the long saphenous and the perforator veins possess incompetent valves must be true. It should be added, however, that the entire deep venous system is without competent valves. In no other way could the blood from the abdominal reservoir refill the veins so quickly.

The tourniquet is occasionally employed in examining the short saphenous vein. This procedure consists of applying the tourniquet just above the knee when the leg is elevated. If, when the patient assumes the erect position, the leg veins fill rather rapidly, the short saphenous vein is deemed to be incompetent. In such cases it is suspected that some valves in the popliteal vein above the saphenopopliteal junction are abnormal. However, if rapid filling does not occur, the test does not negate the possibility that the vein is incompetent. For this reason, the tourniquet tests are seldom used to test the short saphenous. Instead, while the patient is standing with the knee slightly flexed, the vein is felt and a percussion wave is sought. If on palpation or percussion the vein appears large, the short saphenous should be eradicated. It must be remembered that a large short saphenous vein (W in Fig. 1) may continue proximally and receive its retrograde flow of blood from the long saphenous either near the saphenofemoral junction or at other sites in the thigh below that point.

OPERATIVE PROCEDURE

The patient is examined for systemic diseases and especially for arteriosclerosis, postphlebitic sequelae, arteriovenous fistulas and nevi. A careful history is taken. In 97 per cent of over 5000 patients, it was found that one or both parents had varicose veins. Rarely was it necessary to find this factor in the grandparents. When questioned concerning the presence of varices in their parents, patients often gave an unequivocal "No." One further question, "Have you seen your father's or mother's limbs?" yielded valuable information. For instance, answers such as "No, my mother died when I was an infant" or "I have not seen my father's legs, but he does not complain about them" revealed that many original "no" answers were in fact "don't know" answers. In arriving at the figure of 97 per cent these "don't know" answers were discarded and this greatly affected the statistics.

Further examinations concerning the presence of dermatophytosis, eczema, edema, ulcerations, vulval varices, varicocele and hemorrhoids, and the placement of the veins are made. Routinely the only tourniquet test employed is the application of the tourniquet around the thigh. If

only slow filling of the veins in the leg occurs when the patient assumes the erect position, it is believed the patient does not possess post-thrombotic sequelae or arteriovenous fistulas.

Much importance is placed on inspection and palpation of the limbs. Large venous bulbs and, especially, defects in the fascia are sought since these abnormalities are usually the sites of incompetent perforators.[6] These sites and the veins are marked with a weak silver nitrate solution prior to operation. Patch testing for silver nitrate and antiseptics used in surgery is done.

General anesthesia or, in rare instances, local analgesia is employed. If the short saphenous is to be excised, the operation is started with the patient in the prone position since the patient is less disturbed when turned from the prone to the supine position than from the supine to the prone. The patient's head is lower than his feet so that blood is drained from the lower extremity. In treating the short saphenous vein, a transverse incision is made in the popliteal space. The popliteal vein is widely exposed and the short saphenous vein is ligated at its exact junction with the popliteal vein. If such a union does not exist, the saphenous vein is followed deep within the popliteal fossa and there ligated and transected. A search is made for other veins that might make connections between the deep and superficial veins. If the above mentioned W vein (Fig. 1) exists, it is followed to its junction with the long saphenous vein. The perforators in the calf are sought and these are ligated and transected deep within the calf muscles.

The patient is then turned over and a thorough high ligation of the long saphenous is performed. Most patients possess double long saphenous veins and most of the tributaries of the upper end of the saphenous system arise from vein A (Fig. 1). If the ligation is performed at a point indicated by line M (Fig. 1), the main long saphenous vein B will be left intact and the operative procedure will not accomplish its purpose.

The femoral vein is widely exposed and dissections are made well above and below the saphenofemoral junction.[3] Any of the tributaries that normally connect with the proximal end of the saphenous vein may instead make direct union with the femoral vein. All such veins are ligated at their exact junction with the femoral vein. Both the saphenofemoral junction and the bifurcation of the common femoral artery lie about 3.5 cm. below the inguinal ligament. If the profunda femoris artery branches from the medial side of the femoral artery instead of the usual lateral side, it may cross anterior to the femoral vein and care must be taken not to injure it.

Next the saphenous and perforator veins in the thigh and lower leg are excised. This procedure was detailed in previous articles[5, 6] and a somewhat more simplified description will be given here. Incompetent perforators in the thigh are relatively rare, but if present they are eliminated. A large bulb at point C (Fig. 1) often exists but it does not necessarily represent an incompetent perforator. This bulbous varix is most often caused by

cross currents of blood that meet at point C. The saphenous vein B′ is protected by the deep fascia overlying it, and as its flow of blood meets that in the much less well-protected vein B‴, a bulb is often formed. An incision is made over the bulb and the area is explored for a possible perforator. Rarely, a medial thigh perforator Q vein (Fig. 1) exists which does not make direct connections with veins B′ or B″. If present it is ligated and transected deeply.

Next, the incompetent perforators and varices in the leg are excised.[3, 6] An oblique incision is made on the inner aspect of the leg just distal to the knee and the veins B″ and B‴ (Fig. 2) which were dissected from the thigh are exposed. From either or both of these veins, tributaries may course anteriorly or posteriorly. The vein that courses posteriorly often unites with the short saphenous in the midcalf area and incompetent perforators that extend deep into the calf muscles may exist along its route. If present, they are dissected deeply into the muscle where they are ligated and transected. Often the vein that courses anteriorly over the tibia is very tortuous, and, if so, it should be excised by dissection; it is large and brittle and a stripper cannot be used.[4]

Perforator sites that were previously marked are then diligently explored in the following way. A longitudinal incision, 3 cm. in length, is made over these sites and vein B″ is picked up. Subcutaneous tissue is stripped away from the deep fascia and either a weakness in the fascia can be felt or the perforator is located by hooking the index finger around it as it emerges from the deep fascia. The perforator is followed deeply beneath the crural fascia in order to eliminate the possibility of missing a Q vein (Fig. 4). In Figure 4, two perforators are seen emerging from beneath the deep fascia. If the ligation is performed at the indicated incorrect site, the reflux of blood will be through the accessory perforator Q vein and the procedure will fail to accomplish its purpose.

After previously marked perforators are excised, vein B″ is grasped and examined for other possible perforators. Figure 2 details the placement of these perforators as being 40, 35, 30, 24, 18.5 and 13.5 cm. respectively above the sole of the foot. Vein B″ is followed distally, stripped digitally and with a stripper, and if another perforator is found it is excised. Incompetent perforating veins that were not suspected prior to the operation are often found by this method.

The perforators on the lateral aspect of the leg should be explored in the same manner. Although not common, incompetent perforators may occur on the lateral or posterior aspect of the thigh. If present, they should be ligated at a point deep beneath the fascia lata, if possible, at their junction with a major vessel of the deep venous system.

Following operation the lower extremity is wrapped from the groin to the ankle in roller gauze, and adhesive or ace bandages are snugly applied in order to prevent swelling. The operative time for each extremity is approximately two and one-half hours and when two extremities are being

treated it is desirable to have the aid of at least one additional surgeon. Teamwork is very helpful.

The futility of the intramural stripping procedure as often practiced can be demonstrated if we return to Figure 2. If vein B″ is eliminated from the drawing, many collateral and accessory perforator Q veins are left intact. The use of this type of stripper as advocated by Myers, however, is a different matter.[2, 4] He has included in his method all the fine points necessary to eliminate incompetent perforators and has also diminished the operative time.

Over 2700 radical surgical dissections of varicose veins in the lower extremities have been done at the University of California Medical Center. In the entire group no deaths or massive hematomas have occurred. Side effects were mild. All patients were walking within 28 hours after operation. Ace bandages were worn for about six weeks. There was a 97.5 per cent improvement. Patients possessing vulval varices were not included in these statistics. It was seldom necessary to give sclerosing solution postoperatively. I do not believe that any operative procedure is useful in postthrombotic patients, except possibly in those who have had an extremely mild phlebosclerosis.

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Surgical Anatomy in the Treatment of Venous Stasis

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Undoubtedly varicose veins have plagued man since time immemorial. Yet the fact remains that the surgical treatment of varicose veins and venous stasis still has a bad reputation. The chief reasons for this failure are that recognition of the pathological anatomy and its correction have been inadequate.

Many surgeons fail to appreciate that there are three systems of veins in the lower limb. These are (1) the superficial or saphenous veins, (2) the deep veins and (3) the perforating veins which carry blood from the superficial into the deep system by perforating the deep fascia. All of these systems have valves which insure that the direction of flow of the blood, under the action of the "peripheral heart," is always upward and inward from the superficial to the deep system. Venous stasis will result if the valves in any one or all of these systems are incompetent.

Incompetency of the superficial system, that is, simple, primary varicose veins, will not produce severe chronic venous stasis. In fact, it will not produce more than a feeling of heavy fatigue, mild aching, occasionally some swelling, some spotty pigmentation after many years, occasionally a patch of dermatitis and very rarely a small, acute, superficial ulcer which will heal readily with elastic support or elevation of the limb.

When the deep system has been damaged by phlebitis we see the severe chronic venous insufficiency characteristic of the postphlebitic syndrome. Here there is incompetence of all three systems and the peripheral heart cannot function when its valves have been destroyed.

Veins of the perforating system can become incompetent with or without a history of deep phlebitis. Their valves can be destroyed by trauma, by superficial phlebitis extending into the perforating vein but not reaching the deep vein, or by the process of phlebosclerosis which wrinkles the valve cusps and renders them incompetent. When a perforator becomes

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incompetent, each muscular contraction of the leg will force blood out of a portion of the deep vein, between two of its competent valves, through the leaking perforator to the superficial system. When incompetent perforating veins are added to simple varicose veins, varying degrees of chronic venous stasis will develop in proportion to the number of incompetent perforators present. The symptoms will vary from a marked full feeling or a bursting pain in the calf to those of the most severe degree of chronic venous stasis with pain, swelling, induration, subcutaneous fibrosis, pigmentation, severe dermatitis and chronic ulceration.

Through the years various authors have stressed the importance of the perforating veins. Homans[3] was one of the first to do so in 1917. No one, however, suggested a logical method of dealing with these veins until Linton[5] in 1953 described his fasciectomy, which consisted of a subfascial ligation of the perforators and excision of the deep fascia of the posterior compartment of the leg. In 1949, Sherman[7] made an outstanding contribution by describing in detail the anatomical distribution of the perforating veins. In 1953, Cockett and Jones[2] suggested a less radical operation than Linton's consisting of a 6- to 8-inch incision on the medial aspect of the lower leg and ligation of the perforators found deep to the fascia in this area.

The object of this paper is to describe the normal and pathologic anatomy of the veins of the lower extremity. Based on this knowledge, a radical method of treatment of chronic venous insufficiency, which has been very successful in over 500 cases, will be described.

SURGICAL ANATOMY

Fasciae of the Lower Limb

The *superficial fascia* is of more physiological than surgical importance. It consists of a superficial fatty layer and a deeper membranous layer. The main stem of the long saphenous vein is covered by the deeper membranous layer but the tributaries of the saphenous vein lie in the superficial fatty layer. This accounts for the prominent bulging of varicose tributaries while the incompetent main stem is not apparent, except in a thin leg. The short saphenous vein is enclosed in an envelope of thicker fascia in the upper two-thirds of its course. The deep layer of this envelope is the traditional deep fascia and the superficial layer is either a thicker layer of superficial fascia or a superficial layer of the deep fascia. The short saphenous pierces the deep fascia just below the flexor crease of the knee to pass into the popliteal fossa. Gross incompetence of the main short saphenous vein is not clinically evident owing to this fascial envelope. Rather, varicosities are seen on the lateral side of the leg and in its lower third behind the fibula and external malleolus where the short saphenous and its tributaries are superficial to the deep fascia.

The fascial layers overlying the main stems of the long and short saphenous veins are physiologically important because they are components of the peripheral heart or pump; muscular activity of the normal limb causes these fascial layers to compress the saphenous veins and to force blood upward through the competent valves.

The traditional *deep fascia* is a dense fibrous membrane investing the entire lower limb like an elastic stocking. It is a very important part of the peripheral pump because it resists the increase of pressure of blood in the deep veins and of tissue fluid that tends to collect in the dependent part under the influence of gravity. The deep fascia is thin over the gastrocnemius, but on the anterior and fibular aspects of the leg, more especially in its proximal half, it is very strong and dense, and here gives attachment to the superficial fibers of the muscles that it covers.

There is only one large opening in the deep fascia of a normal limb, namely, the fossa ovalis, and this is protected by the cribriform fascia. The long saphenous vein pierces the cribriform fascia to join the common femoral vein. There are many other smaller openings in the deep fascia through which perforating veins pass more or less obliquely.[1] The anatomical distribution of the perforating veins is relatively constant because most of them pass along intermuscular septa to reach the deep veins and this determines the sites at which they must perforate the fascia.

Intermuscular Septa and Perforating Veins

In the Thigh. Three *intermuscular septa* pass from the investing deep fascia to the linea aspera of the femur, forming three compartments for the extensor, the adductor and the flexor muscle groups. The medial septum separates the extensors and the adductors and helps to form the walls of Hunter's canal and reaches the surface along the medial margin of the sartorius. The lateral septum separates the extensors and the flexors and is attached to the investing fascia along the whole length of the iliotibial band. The posterior or posteromedial septum, separating the flexors and the adductors, is not as important as the first two from the point of view of perforating veins.

There are two constant *perforating veins* in the thigh, both related to the medial intermuscular septum. The most important is the mid-Hunter's canal perforator which connects the long saphenous to the superficial femoral vein by piercing the deep fascia at the medial border of the sartorius. (The "superficial femoral" vein is actually the femoral vein but will always be referred to as the superficial femoral vein in contradistinction to the profunda femoris or "deep femoral" vein.) The second perforator pierces the deep fascia at the junction of the middle and lower thirds of the medial aspect of the thigh and passes behind the sartorius to connect the long saphenous vein with the subsartorial plexus of veins. The subsartorial plexus connects above with the superficial femoral vein and below with the genicular plexus of veins, the posterior tibial vein by way of the

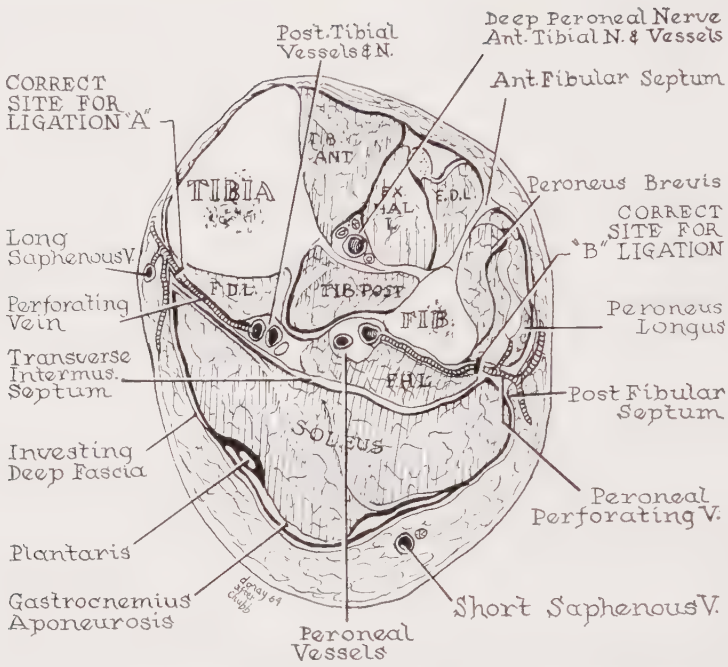

Figure 1. Cross section through the lower part of the middle third of the leg showing the relationship of perforating veins to the transverse intermuscular septum and the posterior fibular septum. The correct sites for ligation of these veins are at "A" and "B," deep or anterior to the septa. The two main tributaries of the peroneal perforator should be individually ligated also.

highest perforating vein at the upper end of the medial border of the tibia, and finally may connect with other perforating veins on the medial aspect of the lower part of the leg.

Small, inconstant perforating veins pierce the iliotibial band to connect with the tributaries of the profunda femoris vein and occasionally one of these may be quite large and become incompetent, and is usually situated in the middle third of the lateral aspect of the thigh. Similarly, an inconstant perforator may be found in the midline of the posterior aspect of the thigh, at the junction of the middle and lower thirds, also connecting with tributaries of the profunda femoris vein.

IN THE LEG. Here, again, are three *intermuscular septa,* in addition to the interosseous membrane. They are the anterior and posterior fibular septa and the transverse intermuscular septum (Fig. 1). The two former are continuous with the deep fascia superficially, and are attached to the anterolateral and posterolateral borders of the fibula respectively, thus forming a fibro-osseous compartment containing the peroneal muscles. The transverse intermuscular septum occupies the posterior compartment of the leg and lies between the superficial flexors (gastrocnemius and soleus), and the deep flexor muscles. On the medial side of the leg this septum is

attached in its upper part along the soleal line of the tibia, since it is deep to the soleus, and does not fuse with the investing deep fascia until it can reach the medial border of the tibia at about the junction of the middle and lower thirds where the soleus ceases to take origin from the tibia (Fig. 2). From this point downward the fused transverse septum and investing fascia form an extremely dense layer which goes on to form the flexor retinaculum designed to maintain the position of the strong tendons passing from leg to foot (tibialis posticus, flexor digitorum longus and flexor hallucis longus). Of great surgical importance is this line of fusion of the transverse septum and the investing fascia. After the two layers meet at the junction of the middle and lower thirds of the medial border of the tibia, the line of fusion inclines posteriorly and distally until it reaches the upper border of the calcaneus anterior to the insertion of the Achilles tendon. The posterior tibial artery and its venae comites lie deep to the transverse intermuscular septum and pass under approximately the midpoint of the oblique line of fusion of the fascial layers and continue on deep to the flexor retinaculum behind the medial malleolus to reach the foot (Fig. 2).

There are 16 constant *perforating veins* in the leg which may become incompetent. Eight of these drain into the posterior tibial vein, four into the peroneal vein and four into the gastrocnemius and soleal veins. Other perforating veins pierce the dense fascia of the anterior compartment to connect with the anterior tibial vein but these seldom become incompetent, probably because they are very small and are supported throughout their

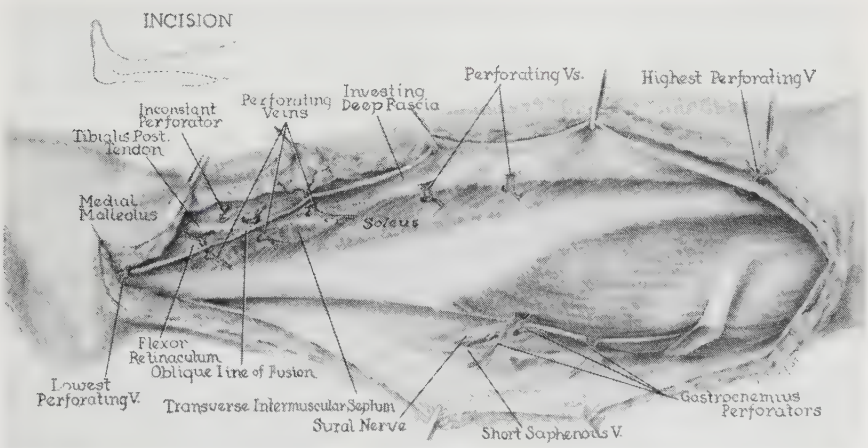

Figure 2. Drawing from a cadaver of the fasciotomy procedure showing the exposure obtained of the perforating veins which connect with the posterior tibial vein. The incision in the investing deep fascia follows the oblique line of fusion of this fascia with the transverse intermuscular septum distally along the upper border of the flexor retinaculum. During the actual operation forceps are never used for retraction as shown on this cadaver.

subfascial course by the muscles which take origin from the deep surface of the fascia.

The perforating veins related to the *posterior tibial vein* are on the medial side of the leg (Fig. 2). The lowest perforator lies close to the upper surface of the os calcis in the fibrofatty tissue anterior to the medial border of the Achilles tendon, and pierces the flexor retinaculum to reach the deep vein. Three important perforators lie in the oblique line of fusion of the transverse septum and the investing deep fascia. One is in the lower part of this line about 4 cm. above the upper surface of the os calcis; the second is at the midpoint of the line exactly where the posterior tibial vein passes deep to the line; the third is at the upper end of the line of fusion of the fasciae where it commences at the medial border of the tibia. The perforator in the middle of the line of fusion is the most commonly incompetent perforator in the leg and it connects superficial veins directly with the posterior tibial vein, having no direct communication with the long saphenous; it is the perforator usually responsible for the development of chronic ulcers in the customary position above and behind the medial malleolus. The perforator at the upper end of the line of fusion is the next most commonly incompetent perforator; it usually has two or more tributary branches draining superficial veins in the area, one of these will be quite short and communicates with the long saphenous vein. Stripping of the long saphenous will destroy this one communicating tributary but will not eradicate the perforating vein proper. Above this level the three more perforators present are closely applied to the medial border of the tibia at about 5-cm. intervals. All of these have several tributaries from superficial veins, only one of which will connect with the long saphenous, and the stem of the perforator is held against the bone by the fascia which usually has to be incised to properly expose the vein passing deeply to reach the posterior tibial vein; they may also receive a branch from the soleus (Fig. 1). The highest of these perforators connects also with the subsartorial vein. One other perforator connecting with the posterior tibial vein must be described. It pierces the flexor retinaculum about 3 fingerbreadths above the lower end of the medial malleolus and passes between the tendon of the tibialis posticus and the tibia (Fig. 2). All of these perforators are accompanied by a small artery from the posterior tibial.

The four perforating veins related to the *peroneal vein* are on the lateral side of the leg along the line of the posterior margin of the fibula. The lowest is situated about 5 cm. above the posterior upper surface of the os calcis and the others are found at 5- to 8-cm. intervals above that. They drain the superficial veins in this area and pierce the investing deep fascia along the line of attachment to it of the posterior fibular septum (Fig. 1). The main stem of the perforator passes inward on the anterior surface of, or actually in the posterior fibular septum, and picks up one or more tributaries from the peroneal muscles and the soleus; it then passes deep to the flexor hallucis longus on the posterior surface of the fibula and

joins the peroneal vein which is lying close to the medial margin of the fibula. The transverse intermuscular septum fuses with the posterior fibular septum and the dense layer thus formed goes on to its attachment with the investing deep fascia; for this reason these perforating veins are not easily seen from the posterior aspect of the posterior fibular septum. Again, all of these perforators are accompanied by small arteries which are branches of the peroneal artery.

The four perforators which drain into the *gastrocnemius* and *soleal veins* differ from those previously described in that they do not pass along intermuscular septa. Two are on the medial half of the calf and one on the lateral half and after piercing the investing deep fascia they may lie for 2 to 5 cm. on the surface of the respective bellies of the gastrocnemius before entering that muscle to join deeper veins which eventually terminate in the popliteal vein (Fig. 2). These perforators drain superficial veins on the posterior aspect of the calf which communicate with tributaries of the long and short saphenous veins. During their long subfascial courses, they may receive other tributaries on the surface of the muscle. The fourth perforator of this group forms a direct communication between the short saphenous and the gastrocnemius veins and accompanies the sural nerve wherever the latter pierces the deep fascia, usually about the middle of the leg.

In the Foot. There are four perforating veins in the foot that are occasionally of surgical importance. One is situated about 2.5 cm. below the inferior tip of the medial malleolus and a second is about 3.5 cm. below and anterior to the anterior margin of the medial malleolus. The other two are on an arc about 3 cm. anterior and below the external malleolus.

DIAGNOSIS

Determination of which systems of veins are incompetent is obviously a basic prerequisite of adequate surgical treatment. A detailed history must be obtained of the onset of the "varicose condition," of the occurrence of superficial or deep phlebitis related to pregnancy, surgery or systemic illness, of severe contusions producing gross swelling of a leg or of acute swelling requiring removal of a cast from a fractured leg. Careful inquiry will differentiate between superficial and deep phlebitis and venograms will not be necessary to decide what treatment is required. Physical examination will readily reveal incompetency of the perforating veins of the leg. The signs and symptoms of incompetency of the various systems were mentioned above and will be alluded to when discussing treatment.

Trendelenburg Test: Method and Interpretation

To obtain objective evidence of the incompetency of the saphenous veins and the various perforating veins, the Trendelenburg test will be

found to be almost infallible if properly performed. Soft rubber tubing, ¼ inch in diameter, should be used as a tourniquet in preference to a Penrose drain which cuts into the leg and is painful. An assistant is necessary in order to attain accuracy of timing and of palpation during the test. The examining table, on which the patient lies supine while the limb is elevated and the tourniquet applied, should be of such a height that the patient can swing around and easily stand on a chair without much muscular effort. The chair seat should be of wood or metal so that additional muscular activity is not required to maintain balance as this can influence the filling of veins and also make accurate palpation difficult. The examiner must sit on a stool so that the legs are at eye level and palpation is comfortably carried out.

To carry out the test the patient lies on the table and the assistant elevates the leg to 45 degrees by supporting the heel. The examiner milks the veins free of blood and applies the tourniquet around the upper third of the thigh sufficiently tightly to obliterate the superficial veins but not the deep veins, and the patient immediately stands. From the moment of standing the examiner carefully observes and palpates the varicose veins and at exactly 20 seconds the tourniquet is released. The tourniquet should be released quickly by the assistant, so that the examiner's finger tips can remain on the veins and can sense the impulse or increase of tension in the veins when the tourniquet is released.

The test is called *singly positive* when the veins remain empty during the 20 seconds and then fill rapidly from above on release of the tourniquet. This implies that the long saphenous vein alone is incompetent.

It is called *doubly positive* when the veins below the tourniquet become moderately filled during the 20 seconds and then become more distended on release of the tourniquet. The filling of the varicose veins during the 20 seconds means that they are being fed by an incompetent vein below the level of the tourniquet and this may be the short saphenous or a perforating vein; the rapidity and degree of this filling should be noted. The additional filling on release of the tourniquet is of course due to incompetency of the long saphenous.

When the test is doubly positive with the tourniquet around the upper third of the thigh, further tests will be necessary to determine the situation of the one or more incompetent veins below this level. Therefore, the tourniquet is next applied just above the patella. If this test is singly positive it means that no incompetent veins are present below the knee but that a thigh perforator is incompetent as well as the long saphenous. If this test is doubly positive and the rate of filling is unchanged, there is no incompetent thigh perforator; but if the rate of filling is definitely slower it means that an incompetent thigh perforator exists, which has now been eliminated by the lower position of the tourniquet, and that another incompetent vein is present below the knee.

If the test is doubly positive immediately above the patella, it must

be repeated applying the tourniquet just below the knee. If a singly positive test is now obtained, it means that the short saphenous vein is incompetent. If a doubly positive test is obtained, it means that incompetent perforators are present lower in the leg; a decrease in the rate of filling will indicate that the short saphenous is also incompetent, but this is practically always true when incompetent leg perforators are present.

The Trendelenburg test will not tell us whether the deep vein is incompetent because incompetency of the perforating veins may or may not be accompanied by deep vein incompetency. The decision as to whether the deep vein is incompetent can be made from the history by carefully tracing the usual phases in the development of the typical postphlebitic syndrome.

For the sake of clarity it should be pointed out that the Trendelenburg test is said to be *negative* if the long saphenous vein moderately fills during the 20 seconds and does not become more tense or becomes even less tense on release of the tourniquet. Obviously, the valves of the vein must be competent in order to produce these findings. Such a test may also be obtained during the first few months following a deep phlebitis, while the deep vein is still occluded and the return flow is entirely through the saphenous and collateral veins. A careful history will avoid misinterpretation of such a test.

TREATMENT

Incompetent Saphenous Veins

Stripping of the long and short saphenous veins is the accepted treatment.

Long Saphenous Vein. The fossa ovalis lies 1¼ inches lateral to and 1¼ inches below the pubic tubercle. A 4-inch incision parallel to Poupart's ligament, centered over the fossa, will expose the upper end of the long saphenous vein, the saphenous bulb receiving its customary four tributaries and the saphenofemoral junction (Fig. 3). Identification of the tributaries of the bulb before division of any major vessel will prevent the calamitous error of dividing the common femoral vein or artery. The long saphenous vein should be divided about 2 inches below its termination and the various branches divided and as much of them as can be exposed in the wound excised. The superficial external pudendal vein should be dissected out, if possible, as far as the external inguinal ring, for here it anastomoses with veins in the inguinal canal and labium majus or scrotum. The deep external pudendal vein lies under the deep fascia and may terminate at the saphenofemoral junction or on the medial side of the common femoral vein; if it is seen through the fossa ovalis it should be individually ligated. The saphenofemoral junction is then ligated and transfixed flush with the anterior wall of the common femoral vein; this insures that no blind stump

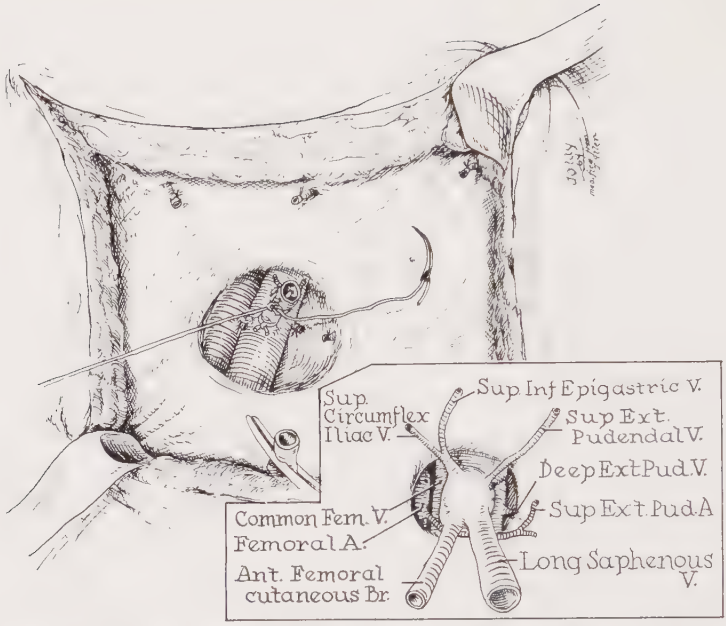

Figure 3. Surgical anatomy of the right saphenofemoral junction and technique of its ligation and transfixion flush with the anterior wall of the common femoral vein.

of the long saphenous is left to harbor a thrombus which might produce an embolus, and also insures that no tributary is left to allow persistent incompetence of the saphenofemoral junction. The superficial external pudendal artery comes out through the fossa ovalis and passes medially usually in the angle between the common femoral vein and the saphenous bulb, but sometimes it crosses the femoral vein proximal to the saphenous bulb. An awareness of the relations of this small artery is helpful in finding the saphenofemoral junction when one is re-exploring a groin where a mass of varicosities has developed following an incomplete saphenofemoral ligation.

The lower end of the long saphenous is then exposed through a 1-inch transverse incision at the level of the anterior prominence of the medial malleolus where the three main tributaries join to form the long saphenous vein proper. Branches of the saphenous nerve should be separated from the vein before its division and ligation of its tributaries. An intraluminal stripper will pass upward more readily than downward, and the vein should be stripped upward to the groin rather than downward to avoid the trauma of pulling toward the lower incision a large mass of stripped vein and fat under the relatively tight skin of the leg.

The anterior femoral branch of the long saphenous is often quite large but too tortuous to allow passage of an intraluminal stripper; however, much of it can frequently be removed by using an extraluminal stripper.

To avoid postoperative hematoma, the foot of the operating table

should be raised 15 degrees during the entire procedure and adequate manual pressure with sponges maintained along the line of stripping until active bleeding has ceased, and finally all blood clots expressed as completely as possible from the whole length of the limb.

Short Saphenous Vein. Incompetency of the short saphenous is frequently overlooked because the vein is enclosed in a tunnel of fascia, as mentioned above. In carrying out the Trendelenburg test for incompetency of this vein, one must frequently use the dilated tributaries behind the external malleolus for the assessment. In approximately 90 per cent of cases of long saphenous incompetence the short saphenous is also incompetent.

After completion of the stripping of the long saphenous vein, the patient is turned into the prone position and the popliteal fossa is opened through a transverse 3-inch incision, placed ½ inch above the flexion crease, incising the popliteal fascia in the line of the incision; the lateral popliteal nerve must be carefully avoided where it lies in the lateral part of the fossa along the posterior border of the biceps femoris. The short saphenous vein, accompanied by the sural nerve, lies between the two heads of the gastrocnemius. Its termination is very variable. It usually ends by joining the popliteal vein and sends a small vein upward into the thigh. It should be ligated and, if very large, transfixed close to the popliteal vein. If it does not enter the popliteal vein, or has only a small communication with it, the short saphenous will continue on its proximal course and connect with a branch of the profunda femoris vein or the inferior gluteal vein, or it may join the long saphenous in the thigh. Under these circumstances the short saphenous should be ligated as high as possible in the popliteal fossa. Its lower end is exposed behind the external malleolus and after ligation of its several tributaries here, it is stripped from the ankle to the popliteal fossa. The sural nerve, lying deep to the short saphenous behind the external malleolus, is quite large here and must not be mistaken for the vein.

Incompetent Perforating Veins

Incompetence of the perforating veins of the leg added to incompetence of the saphenous veins produces a completely different pathological picture. It may resemble the leg of the full-blown postphlebitic syndrome, yet the patient will deny ever having had gross swelling or any incident necessitating bed rest. The degree of pathological change is directly proportional to the number of incompetent leg perforators; incompetent thigh perforators alone will not produce the pathological changes, but they increase the severity of the symptoms.

The pathognomonic sign of incompetent leg perforators is the presence of subcutaneous fibrosis on the medial aspect of the lower third of the leg. In the early phase in some patients this may develop as an area of tender, brawny induration. After months or years the inflammatory character

disappears to be replaced by typical subcutaneous fibrosis. The subcutaneous fat feels thinner than normal, firm, hard and lumpy; in long-standing cases calcified plaques may be present owing to destruction and saponification of the fat. Varices can be palpated in the area of fibrosis like the holes in cheese.

Many theories have been advanced to explain the development of subcutaneous fibrosis and several factors undoubtedly combine to produce it, such as venous stasis, venous hypertension, anoxia and edema followed by fibrosis. Space does not allow a discussion of these etiological factors. Pigmentation of the overlying skin is due to diapedesis of red blood cells through capillary walls made more permeable by hypertension and anoxia.

The subcutaneous fibrosis does not develop circumferentially around the lower part of the leg with a horizontal upper margin, like water in the bottom of a barrel, but is found in a limited area immediately distal to the offending incompetent perforator. An interesting observation has been that when several incompetent perforators are present, the extensive area of subcutaneous fibrosis will have a serpiginous upper border, each peak of which will indicate the site of an incompetent perforator. The tell-tale subcutaneous fibrosis immediately below each perforator resembles the rust mark on a cement wall below a leaking pipe.

Every chronic ulcer is directly related to an incompetent perforator lying deep to it or immediately proximal to it. The fact that the perforators along the upper oblique margin of the flexor retinaculum are the most commonly incompetent explains why the medial lower third of the leg is the usual site for subcutaneous fibrosis, pigmentation, dermatitis and ulceration (Fig. 2). Similar pathological changes about the external malleolus are caused by incompetent peroneal perforators.

It is impossible to eradicate all incompetent perforators by palpation of the windows in the fascia through which they penetrate and then individual ligation through multiple incisions. What is usually thought to be a window in the fascia is merely a varix surrounded by subcutaneous fibrosis. Furthermore, it has been our experience that more perforators than were expected are found to be incompetent.

The only logical method of dealing with the leg perforators is by a procedure which we have called a "fasciotomy." It is a subfascial exploration of the entire posterior compartment of the leg which allows visualization of every perforator and ligation of all those incompetent. Every incompetent perforator must be eradicated, for if even one is left the peripheral pump will still "leak" and the deleterious effects of chronic venous stasis will continue. This procedure is just a modification of Linton's "fasciectomy."[5]

Preoperative elevation of the limb for one or two days to get rid of all edema is important because edematous tissues cannot heal well, and since the skin is already suffering from nutritional deficiencies every precaution must be taken in order to obtain primary healing.

The initial step in the operation is to ligate the saphenofemoral junction but the actual stripping of the long saphenous is not done until the fasciotomy is completed.

The fasciotomy incision is marked on the leg with the knee in extension and the ankle at a right angle (Fig. 2). It commences 1 fingerbreadth below the inferior tip of the medial malleolus, curves upward midway between the posterior border of the medial malleolus and the Achilles tendon, then straight up to the junction of the middle and upper thirds of the leg where it veers posteriorly to end just below the flexion crease of the knee lateral to the tendon of the semitendinosus. With the knee flexed and externally rotated and the ankle at a right angle, the incision is made through skin and subcutaneous fat down to the deep fascia. To control bleeding, which may be considerable, pick up the divided veins with hemostats but do not include fat in the forceps; ligation of these veins is delayed until closure of the wound when they will be dealt with in a specific manner. Near the upper end of the wound, the deep fascia is incised and opened upward and then downward as one would the peritoneum. On reaching the junction of the middle and lower thirds of the leg, the fingers advancing deep to the fascia will encounter the oblique line of fusion of the investing deep fascia and the transverse intermuscular septum, in other words, the oblique upper margin of the flexor retinaculum. The incision of the investing deep fascia is now carried downward and posteriorly along this oblique line, dividing the attachment of the investing fascia to the upper margin of the flexor retinaculum (Fig. 2). The posterior skin flap below the oblique line is shaved off the retinaculum so that the full thickness of subcutaneous fat is left with the skin to help maintain its blood supply. During the course of the dissection along the oblique line, the middle and lower perforators situated here will be encountered and, if incompetent, should be ligated deep to the transverse intermuscular septum on the surface of the flexor digitorum longus; this can be accomplished by enlarging, if necessary, their windows in the fascia. The division of the investing fascia from the flexor retinaculum is continued with scissors down alongside the Achilles tendon until the upper surface of the os calcis is reached. Sometimes the lowest perforating vein can be seen just above the os calcis and can be divided and ligated, but if none can be seen in the fat a deliberate attempt should be made to cut any vein in this area and control the bleeding by packing. It is a more serious error to leave an incompetent perforator here than to divide a normal one.

The anterior flap of skin and subcutaneous fat is lifted off the upper part of the oblique line until the perforator at the upper end near the medial border of the tibia is reached. The investing fascia need not be divided along the line of fusion here. The tributaries of this perforator can be ligated superficial to the retinaculum, and the main stem of the perforator can be ligated through its window deep to the transverse intermuscular septum. Before leaving this part of the leg the surgeon should

Figure 4. Fasciotomy procedure on left leg showing retraction forward of gastrocnemius and Achilles tendon to expose peroneal perforating veins.

shave the anterior flap of skin and fat from the flexor retinaculum just in the midportion between the medial malleolus and the upper border of the retinaculum, in order to visualize the perforator situated between the tibialis posticus tendon and the medial border of the tibia 3 fingerbreadths above the tip of the medial malleolus. This perforator is very inconstant in size and, if very small, its position will be recognized only by the division of its accompanying arterial twig (Fig. 2).

The upper two-thirds of the anterior skin flap, with its deep fascia, is now lifted forward and with a few touches of the knife the attachment of the deep fascia to the medial border of the tibia can be divided to expose the three perforating veins clinging closely to the bone and if incompetent they are ligated.

The large posterior flap of investing deep fascia and skin is now swept off the bellies of the gastrocnemius. The two medial gastrocnemius perforators will have to be divided, regardless of their state of incompetency, in order to obtain the required exposure. The proximal ends of these perforators should be tied off in the substance of the muscle so that they will be completely supported by muscle. The perforator accompanying the sural nerve through the fascia is now ligated. The lateral gastrocnemius perforator is the next to be assessed.

The peroneal perforators are now examined by retracting the Achilles tendon and the lower part of the soleus to expose the line of fusion of the transverse intermuscular septum and the posterior fibular septum; the main stems of these perforators lie deep to this line of fusion and can be seen through the fascia (Fig. 4). They can be exposed by incising the fascia

or making a hole through it with a pair of forceps. If the vein is incompetent it should be tied in the substance of the flexor hallucis longus on the posterior aspect of the fibula so that all its tributaries are eliminated.

Experience will help one to decide when a perforator is incompetent. They can normally be the size of a wooden match, but if larger they are probably incompetent. Definite signs of incompetency are gross dilatation to the size of a lead pencil, obvious irregular varicose dilatation, phlebosclerosis producing variations in the thickness of the vein wall or marked thickening and friability of the wall.

The short saphenous vein is now stripped through the fasciotomy wound. The deep fascia is incised alongside the short saphenous vein about a handsbreadth below the popliteal fossa and the vein lifted out of its bed and divided between forceps. The upper portion is removed by cutting the fascia along the course of the vein and with rather strong traction the short saphenous can be pulled down and ligated close to the popliteal vein or higher if it does not enter the latter. The lower end of the short saphenous is exposed by an incision behind the external malleolus and stripped up the leg, protecting the fasciotomy flap from too much trauma by exerting countertraction by thumb and finger closely behind the button of the stripper as it advances; a hematoma must be avoided along the short saphenous tunnel after the stripping.

The lower end of the long saphenous vein is exposed at the medial malleolus and stripped up to the groin. If an incompetent medial thigh perforator was diagnosed it should be exposed through a longitudinal incision and ligated deep to the fascia before the long saphenous is stripped, otherwise hemorrhage in the tissues will make it extremely difficult to find.

The veins originally picked up with hemostats along the fasciotomy wound are now dealt with in a special manner. The healing of the fasciotomy wound is very precarious because of the poor blood supply of the very large posterior flap and also because the skin of the lower third was already suffering nutritional deficiency. Therefore, veins cannot be simply ligated and their ends left to become necrotic along the margins of the wound. They must be dissected back into the fat, a mosquito forceps applied, excess vein excised, and then tied with 000 plain catgut. A vein running longitudinally on the face of the wound must be excised because it will thrombose and the wound will open at this site about the ninth or tenth day. On the other hand, dissection between the skin and deep fascia to remove a cluster of varicosities which seems conveniently close is not advisable, because this will usually result in necrosis of the overlying skin. Retractors should never be used on the skin flaps, nor should finger pressure on the flaps be maintained for too long in one position for fear of causing thrombosis of small arterial twigs. A rake retractor is permitted under the upper end of the wound only during the removal of the short saphenous vein from the popliteal fossa.

The fasciotomy wound is closed with a running suture of 00 plain

catgut in the investing deep fascia to reconstitute the outer wall of the peripheral pump and to relieve tension on the skin. Interrupted silk sutures are used to obtain accurate approximation of the skin margins. An adhesive elastoplast bandage is applied without tension, over appropriate dressings, from the base of the toes to the upper thigh. Elevation of the legs is maintained on the stretcher leaving the operating room and throughout the entire postoperative stay in hospital.

If *chronic ulceration* is present it is safer to obtain healing by bed rest or pressure dressings before operation. Every ulcer will heal with adequate bed rest and elevation of the legs but if the patient cannot accomplish this at home, hospitalization is necessary and the ulcer should be cleaned up with compresses of Dakin's solution (1:4 strength). It is unnecessary to wait for complete healing of the ulcer; the fasciotomy may be done when the ulcer has changed from an indolent to a healing one and has a beefy red, finely granular granulation tissue base, a narrow blue rim of ingrowing epithelium, no rolled margin indicating infection and no induration or tenderness of the surrounding tissues.[6] The first step in the operation should then be the complete excision of the ulcer by a beveled incision, removing any rim of poor skin surrounding the ulcer and also the underlying, thickened deep fascia. The layer of paratenon overlying muscles and tendons should be left where they are exposed. On completion of the excision of the ulcer, all instruments used for this purpose are discarded. The fasciotomy incision is then made in the usual position upward and downward from the ulcer defect. After the fasciotomy wound has been closed in the usual manner, the defect is covered with a split-thickness skin graft from the thigh and sutured into position. A copious pressure dressing is used and then a very light plaster-of-Paris boot is applied to immobilize completely the ankle and the skin grafted area.

Incompetent Deep Veins

Two to five years after a deep thrombophlebitis the deep vein will have recanalized, producing a valveless vein; the saphenous veins and some of the perforators will also be incompetent. The incompetency of all three systems will produce the typical postphlebitic syndrome during the succeeding years. If just a deep vein of the leg was involved it will be a lower leg syndrome, but if the popliteal, the superficial femoral or the iliofemoral region was thrombosed it will be a whole leg syndrome. In 90 cases, surgical specimens have been obtained from both the superficial femoral and popliteal veins and in all cases there were definite pathologic findings, recanalization being present in both, or only in one and phlebosclerosis in the other, indicating the extensive spread of the inflammatory reaction of the phlebitis along the vein wall.

If the patient gives a typical history of phlegmasia alba dolens and complains of pain and swelling of the thigh, it is obvious that the superficial femoral vein is incompetent. This long valveless tube, creating venous

hypertension in the limb by pressure of the column of blood from the heart to the foot, must be interrupted at the same time as the other two systems are dealt with by stripping and fasciotomy. In the absence of symptoms in the thigh, the deep vein need not be interrupted.

A generous incision is made from the midpoint of Poupart's ligament, passing down lateral to the fossa ovalis, to the apex of Scarpa's triangle. If the anterior femoral branch of the long saphenous vein is encountered it may be divided but the long saphenous proper should not be disturbed at this time. The deep fascia is opened along the medial border of the sartorius and the great vessels exposed. The superficial femoral artery and vein will be found to be adherent to each other as the result of fibrosis following the thrombophlebitis. They must be separated carefully until the upper 2 inches of the superficial femoral vein are completely free and the junction with the profunda femoris vein is clearly demonstrated (Fig. 5). A length of No. 2 silk is passed around the upper end of the superficial femoral vein to be used as a tourniquet. The long saphenous vein is now exposed just below the saphenous bulb, and therefore below its upper tributaries, and another silk tourniquet left here. Manometric pressure readings are now taken in the superficial femoral vein in order to prove its

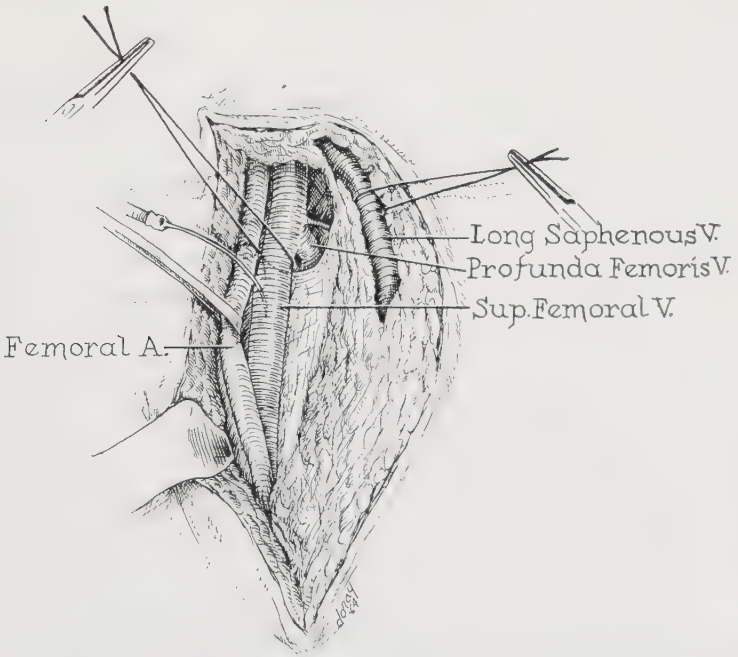

Figure 5. Exposure of the right superficial femoral vein and its junction with the profunda femoris vein. A curved No. 18 needle connected to a manometer has been inserted into the superficial femoral vein and heavy silk tourniquets are in place preparatory to recording the venous pressure on occlusion of the superficial femoral and the long saphenous veins.

incompetence before it is divided.[4] The manometer is filled with 2.5 per cent sodium citrate and connected to a No. 18 needle which is inserted into the superficial femoral vein. The rise in pressure is noted after occlusion of the superficial femoral by tightening the silk tourniquet, and then the additional rise in pressure is noted when the long saphenous is also occluded. In a normal patient the pressure will rise rapidly at least 20 cm. of sodium citrate on occlusion of the superficial femoral and another 20 cm. on occlusion of the long saphenous. In the postphlebitic patient, on occlusion of the superficial femoral the pressure will rise slowly 2 to 6 cm. and another 1 to 2 cm. on occlusion of the long saphenous. This lack of rapid rise on occlusion of the incompetent veins indicates that adequate collateral drainage has developed and that the incompetent veins are serving no useful purpose and can safely be interrupted or eradicated. The highest combined rise in pressure in this series was 15 cm. and it is suggested that if the rise is higher than this the deep vein should not be interrupted.

The superficial femoral vein is now divided, ligated and transfixed flush with its junction with the profunda femoris vein and a portion excised for pathological confirmation of the diagnosis and the distal end also transfixed. The saphenofemoral junction is ligated as previously described. The fasciotomy is then carried out and finally the long saphenous is stripped.

Postoperative Care

The foot of the bed is elevated 8 inches during hospitalization. Quadriceps and ankle exercises are started as soon as possible and almost constant muscular activity is encouraged. Limited ambulation is allowed on the first or second postoperative day following simple stripping but fasciotomy patients must remain in bed until the wound is completely healed. The elastoplast dressing is removed on the seventh day and all or most of the fasciotomy sutures removed and a tensor bandage applied to below the knee. Some degree of delayed healing requiring a few more days in bed is not unusual, occurring in 8.3 per cent of this series of 529 fasciotomies. However, only 11 cases required skin grafts of necrotic areas which developed on the margin of the posterior flap of skin. Small areas of necrosis can take an inordinately long time to heal because the underlying deep fascia is also necrotic; this is recognized by a rim of edema surrounding the area of minor skin necrosis and complete excision of skin and fascia with grafting of the area will be the best treatment.

When ambulatory the patient must wear an elastic bandage or preferably an elastic stocking until all swelling has disappeared; patients are warned to control the swelling by frequent elevation of the limb. Varying degrees of swelling follow such an extensive operation but it is proportional to the amount of preoperative swelling. It decreases rapidly during the first month and may disappear completely during the subsequent months. More than half of the patients will always have some slight swelling toward

evening or after long standing but they will be free of pain. About one month after operation a course of injections should be started to sclerose all remaining superficial varicosities because they are not essential, but rather detrimental, to the collateral circulation. If a patch of dermatitis or superficial ulceration threatens to recur, the region immediately above such an area should be searched for a patent varicose vein feeding this area and obliterated by injection.

During the 15 years in which these cases have been treated the interesting observation has been that, with reasonable care by the patient, these limbs have continued to improve and have not deteriorated with the passage of time.

SUMMARY

It is important to appreciate the role played by incompetence of each of the three systems of veins of the lower limb in producing venous stasis. Of the superficial or saphenous veins the part played by the long vein is well appreciated. The short saphenous is commonly ignored but it has often been found varicose and the cause of symptoms. The perforating veins of the leg, when incompetent, produce marked pathological changes and every chronic ulcer is directly related to one or more of them. Recanalization after phlegmasia alba dolens results in incompetence of all three systems and the development of the typical postphlebitic syndrome.

There are 20 perforating veins of surgical importance below the knee. They are constant in position because they are related to fascial septa. Because of their number and the difficulty in palpating the windows through which they penetrate the deep fascia, it is impossible to deal with them individually through multiple incisions. A radical approach by a "fasciotomy" is necessary to visualize all of them and eradicate every incompetent perforator.

The normal and pathologic anatomy has been described in conjunction with important points of the surgical technique learned during 15 years' experience with 529 fasciotomies. The surgical procedures described have been most successful in relieving the misery suffered by patients with chronic venous insufficiency.

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